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Part I.

Original Contributions.

A PHYSICIAN'S NOTES ON OPHTHALMOLOGY.—CASES OF DISEASE OF THE NERVOUS SYSTEM IN WHICH THERE WERE DEFECTS OF SMELL, SIGHT, AND HEARING.

(A continuation of a paper, p. 78, vol. v.)

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It will, I fear, strike the reader, that in the consideration of Cases XIX. and XX., I speak too much of some symptoms which are out of the scope of a Journal of Ophthalmology. However, much of the interest of a case lies in the number of symptoms which can be put together in an intelligible order. Amaurosis may be studied at once with too much intensity and too little breadth. It is to the Ophthalmologist a Disease of so great importance, calling for particular action on his part, that he may underrate its significance as a Symptom in general conditions of the System. But it is when it occurs with other striking phe-

nomena that we are most likely to discover what optic neuritis means.*

In no department of Medical Science has so much progress been made as in Ophthalmology. An earnest desire that those in more general practice should share in working at the wide questions raised by the discoveries of Helmholtz, Donders, Graefe, and many others in this country, induces me to write in this Journal. In short my object in writing is to urge Physicians and Ophthalmic surgeons to do more work together.

If Amaurosis and Defect of Speech are found together, as in Case XII. and Case XXII., both the symptoms must be studied by one person. Again, I scarcely see how the Ophthalmologist can confine his thoughts to a single sense apparatus, however much his practice may be limited to the treatment of defects of that one. The Physician's wider field of work will not permit a consideration of one of them only. To him defect of Smell has a significance equal to that which defect of Sight has, although the former is of smaller importance. It is obvious that a consideration of the presence of defects of some of the special senses with disease of the hemisphere, and of the absence of striking defect of hearing,† is a very important one to ophthalmologists, physicians, and psychologists, however these differences may be explained. Optic neuritis is the sense disorder which most frequently occurs with one-sided (limb) symptoms. Defect of each of the special senses ought to be especially considered, positively and negatively, in relation to these one-sided symptoms.‡

* When I use the term amaurosis, without qualification, optic neuritis, or the atrophy following it, is meant.

† I have, Lond. Hosp. Reports, vol. i, related a case of hemiplegia from *Disease of the Pons Varolii*, in which case there was deafness on the side of the facial paralysis. When I use the word hemiplegia in this paper, however, I mean the common form of it. I have spoken of this symptom at great length in two lectures in the London Hospital Reports, vol. ii.

‡ More generally, we ought never to forget that the region of the brain of which the corpus striatum forms part, is the most vascular one of the

Hemiplegia, with its allies, therefore, is the extra-ophthalmological symptom which chiefly deserves the consideration of ophthalmologists. Let me now speak even more widely of ophthalmological studies than a view from my own stand-point of practice would permit.

Odd as it may at first sight seem, I feel more and more convinced that there are important reasons why ophthalmologists and those physicians practising in insanity should do much work in common. Not because defects of sight are particularly associated with mental symptoms, nor because insane people may be liable to defects of sight. It is because, I think the special study of the Evolution of Movement [and of Sensation] is best begun at Ophthalmic Hospitals. And so far as we can know anything definite of mind, it is, I suppose, made up of sensory and motor phenomena—the functions of a series of anatomical possibilities in the cerebrum in correspondence with its wide environment, as well as in the spinal cord with its narrow environment.

Any one who has studied the striking physico-psychical symptoms which sometimes occur with paralysis of the limbs of one side—most frequently of the right side—must have been struck by the association of defects of certain highly complex motions, *e.g.*, of articulation, with what are arbitrarily distinguished as mental defects. There is indeed to be traced, as I think, a series of defects continuous in the differentiations of motion, from difficulty of articula-

higher divisions of the nervous system, and the one most often damaged. This is true as a mere clinical observation, and the fact is significant in the study of the relations of vascularity to function, and its consideration is a highly important one in our attempts to fix the seats of certain "faculties." We should contrast optic neuritis, the sense disorder which occurs with one-sided loss of power over our limbs as voluntary organs, with the slow atrophy, &c., which occurs with double-sided symptoms, as disordered locomotion, *e.g.*, "Locomotor Ataxy." We shall thus, I think, learn something of value as to the relations of the special senses in health to voluntary and to involuntary motion, notwithstanding that the association of the *symptom* double amaurosis with one-sided *symptoms* may be an accidental association.

tion to incoherence. It is scarcely necessary to say that the essence of speech is not noise, but motion. Let us (See p. 306) instance a commoner set of motor phenomena.

The muscular irregularities in chorea are, clearly, psychico-physical disorders of motion. For they are a profusion of purposive movements, and not a mere set of spasms or cramps. The variations of muscular life in time are best studied in cases of chorea, epilepsy, &c.

To establish the laws of healthy muscular movements, and of those variations in them which occur in certain altered states of nerve tissue and nervous organs, is a thing of fundamental importance for the progress of the Medical Physiology of the Nervous System. I have no faith that this sort of physiology will make steady progress until the body medical is more highly organised. There is great differentiation in Medical Practice, but not enough unity of aim in cultivators of Medical Science. Now nowhere could we better begin to study *intelligent* movements of muscles than at Ophthalmic Hospitals.

The strange delusional effect produced by paralysis of the superior oblique muscle must have long struck psychologists. Nor does such a delusion lose its significance, however sharply we may define its cause to be merely a loss of a particular way of moving or of helping to move one of the eyeballs. The way a man moves and carries himself who has paralysis of this one ocular muscle, is an excellent illustration of the tendency there is in organisms to equilibration.

The study of the higher movements, as for words, will, I feel sure, derive great help from the Laws of muscular motion, which ophthalmologists have been or will be able to establish in their own department of medical practice. They observe phenomena in which motion and sensation are very distinct, and yet quite inseparable. Were the positive disorders of muscular movements studied in our particular cases of disease of the Nervous System, rather than cases of Strabismus, Chorea, Aphasia, Epi-

lepsy, Locomotor Ataxy, &c., we should, I think, make far more progress than we now do.

Were I free to work in this metropolis at the Physiology of Movement, from gross muscular movements towards thought, in the way I should prefer—supposing to begin with an ordinary general medical and scientific knowledge—I should study first in the wards and in the out-patient room of a General Hospital, such symptoms as Hemiplegia, Chorea, and Epilepsy; next, at an Ophthalmic Hospital Paralysis of the Cranial Nerves and defects of Sight. Next, at a special Hospital, for such symptoms as those of Locomotor Ataxy, and certain Epileptiform Seizures. Then at an Orthopœdic Hospital for Infantile Paralysis and for so-called Contractures. Now I would study specially deviations from healthy movement of the eye-balls, and the rudimentary delusional effects they cause. I would, after this work, return to the General Hospital, to study defects of Speech, Delirium, Apoplexy, Coma, &c. Varieties of defects of Speech I should compare and contrast from difficulty of articulation to incoherence. At this juncture a careful study of laryngeal and throat diseases would be most desirable.*

Of course if any one were to work at different sections of medical practice, so as merely to add isolated series of facts to one another, he would really make little progress in cultivating his own mind. Such a man would dwell

* The above is a mere sketch. It will be seen that I speak chiefly of phenomena, and for convenience in the illustration, solely of phenomena of motion. I think, indeed, it is far more important in the present state of our medical knowledge to study most what disorders of motion really do occur, and their relations to one another, than to work at certain problems as to the causes and meaning of any arbitrary sets of motor disorders. Oddly enough, many prefer working at or thinking on *causes*, to putting in order—any order—the phenomena occurring under their eyes. We shall not, I submit, arrive at a knowledge of the causes of disease until we have paid more precise attention to a wider range of outward phenomena. Of course the object of studying outward phenomena is to obtain a corresponding knowledge of internal states. (See postscript No. 2, p. 306.)

with exaggeration—hurtful to his own organization of medical knowledge— on amaurosis as a defect of sight, and too little on it as a defect of a highly specialized part of universal sensation. He would not improbably neglect altogether such symptoms as defects of smell, which defects, however, are really quite as significant incidents in the revelations which disease makes of function as defects of sight are. Such a student would be altogether unprepared for the last and the very highest medical study viz., of Insanity. Special work at diseases of the mind should, I feel convinced, be begun only after a large real experience of all the special phenomena of motion and sensation that damage to any parts of the Nervous System does or may give rise to. I say a real experience, as I suppose a collection of numberless facts, however accurately gathered, is not held to be of itself a real experience. Unless a man can put the particular phenomena he himself sees under more general Laws, or unless he tries to do this, he can scarcely be said to know or to be studying a thing in any very valuable sense. The knowledge the ophthalmologist has of muscular disorders of the eye ; the knowledge the physician has of defects of articulation, of chorea, and of epilepsy, and that the psychologist has of incoherence and delusions should aim to be Physiological* Units, each different but each related to a wide common knowledge of such Laws—present or in progress—as those of the Evolution of Sensation and Movement in organisms.

The motorial disorders in several cases I have to relate will enable me to refer again to these points.

To return to my particular task. I am compelled to continue to treat amaurosis very widely, and, I fear, loosely. Indeed the four symptoms loss of sight and smell, defects of taste and of hearing, occur in one patient, as Case XXI. shows. If I find, as in Cases XII. and XXIII., loss of

* Physiological Unit is a term introduced by Herbert Spencer. To understand the wide meaning of this term, Mr. Spencer's work on Biology must be studied.

speech with amaurosis, I must consider the two symptoms together. Therefore in relating and commenting on the observations I bring forward, I shall not dwell more on defects of sight than on any other important symptoms the cases may present for the consideration of cultivators of Medical Science. I am obliged to dwell on symptoms further from amaurosis even than anosmia or loss of speech may seem to be,—on such symptoms, for instance, as epileptiform seizures,—because in my field of work certain convulsive attacks occur not unfrequently with amaurosis. I have no doubt the experience of ophthalmologists on this last point is very different. A physician sees those cases of amaurosis in which convulsions occur, because convulsion is a physician's symptom. And when severe convulsions come on in a patient attending at an Ophthalmic Hospital, he will probably leave for a General Hospital. I have referred to this,—the social aspect of our studies,—in some remarks, p. 65 (see also remarks following Case XXIII.). The following case further illustrates those remarks. I have already published most of the facts of the case in the Medical Times and Gazette, August 30, 1864, pp. 481-2.

See Case XX.

In every disease we have to seek (1) the Tissue affected; (2) the Organ damaged; and (3) the Function disordered. This very rudimentary division of our thoughts on single cases into, as it were, three interweaving lines of thought, constitutes, I conceive, a natural basis for the Classification* of cases of disease. Each case, to keep to

* On these points I have already remarked, partly with reference to hereditary transmission and partly with reference to classification of disease. See Brit. Med. Jour., Nov. 24, 1866. Also London Hospital Reports, vol. i. "Lecture on the Study of Diseases of the Nervous System." The distinctions I make betwixt the life, structure, and function of a part will seem to be artificial. "Disorder of function" is almost a contradictory expression. It will, however, be found, I think, that Units of Function do not coincide with Units of Nutrition (see note on arterial regions), and it is convenient to use

the Nervous System, should not be described from disorder of Function only, as "Locomotor Ataxy" nor from damage to the Organ only, as "Disease of the Cerebellum," nor from Tissue changes alone, as "Syphilitic Disease of the Pia-mater," but, when possible, from all three. And when we can get to know only the disorder of function, the gap in such a classification keeps us in mind where speculations and work—not explanations—are wanted. In Case XX we may, I think, venture to speak on each of the three points, and I shall do so at some length, as, besides the question this individual case particularly raises, *i.e.*, the real nature of so-called Syphilitic Diseases of the Nervous System, it leads us to a more general consideration of the secondary changes with which various sorts of disease in the hemisphere are sometimes associated.

There can be little doubt that there is disease near the right (2) corpus striatum, as in Cases XII, XIII, XVI, XVIII, and XIX. It is most probably disease in the (1) connective tissue of the pia-mater, as in Case XIII. (See further on diffused changes.) The (3) functions disordered are the epileptiform seizures and the blindness.

To speak, first, of the tissue changes. There are two chief ways in which syphilis brings about hemiplegia, and, as I think, unilateral epileptiform seizures also. The patient whose case is now the text had both these symptoms.

(*a*) By sudden obstruction of cerebral arteries already diseased.*

(*b*) Hemiplegia—as I believe happened in Case XX—comes on as a consequence of changes associated with coarse and obvious syphilitic disease of the surface of one hemisphere, which coarse disease may or may not actually

the term *disorder of function* in contrast to that of *harmony of functions*. I intend shortly to consider the whole of these points from the basis of *Classification of Diseases of the Nervous System*."

* I have (Lancet, Oct. 27, 1866) spoken of syphilitic disease of the cerebral arteries. I have there referred to Wilks' and Bristowe's previous researches.

invade the motor tract itself. Under these last circumstances the hemiplegia is nearly always associated with convulsion.

I think it most likely that there is coarse disease in the case I have just related, as there was in Case XIII, rather than mere softening from plugging of the right middle cerebral artery. I think so because the sort of amaurosis the patient had is generally the result of* coarse disease within the head. And, of course, we must judge also by the general symptoms, of which amaurosis is only one, when we can get to know them. I do not judge by the particular kind of convulsive attack, as I believe seizures of this sort occur after softening from plugging of branches of the middle cerebral artery. Indeed, when we get enough knowledge, I hope we shall find that conditions of the optic disc—phenomena which can be more precisely estimated than pain in the head and vomiting—will help us to determine something as to whether the internal changes associated with these convulsive seizures are coarsely organic or not. At present to urge that optic neuritis, with epileptiform seizures, points to coarse disease within the cranium as the cause (indirect) of the seizures, would be to appear to be arguing in a circle. At present I can only say that *most probably* there is coarse local disease in this case, as in Case XIII. (See p. 26.)

I will now digress to allude to the treatment of syphilitic nervous affections, for this will almost at once lead me back to a more precise consideration of the nature of the Tissue changes which give rise to or permit the particular symptoms my patient has had.

* However, since the last paper (p. 78) was printed, I have seen an autopsy made by Dr. Moxon, on the body of a man who had previously been under my care—before that under Mr. Hutchinson's care—for optic neuritis, and in this case no such *coarse* intracranial disease was discovered. (See Ophthalmic Review, April, 1866.) Then there is the source of fallacy that coarse disease of the nervous system is more likely to prove fatal, and thus my observations of cases, *post-mortem* and *ante-mortem*, may not be fit for comparison. I, however, judge by other symptoms, as by vomiting, &c., and by examinations of the eyes in many cases of chorea.

Although I have much faith in the power of iodides and mercurials to reverse the recent changes which syphilis induces in the Tissues, I have not very much hope of cure when treating cases of convulsions, hemiplegia, and amaurosis, the results of syphilitic disease of Nervous Organs. This is an important distinction.

In the first place, it would be as hopeful a task to try to procure the absorption of tubercle as to get some of these syphilitic "deposits" taken up by the drugs I have named. An ophthalmic surgeon would not give drugs in the hope that lymph effused on the iris six months before would disappear. He could only hope for success when he could interpose whilst uproarious changes were actively going on. Similarly the physician can only hope to get rid of lymph effused in the pia-mater when recently effused —as when a patient consults him for severe pain in a limited part on one side of the head; not when, some time later, he comes for a "fit," due to diffused changes in his brain. But if we really could get rid of these "deposits" altogether we should, to cure the patient of his epileptiform seizures, &c., have to reverse also the changes of nutrition of nerve tissue which the deposits had "excited"; for it is probably—I suppose this is the general belief—on secondary changes that the amaurosis and the epileptiform seizures depend.

No doubt the various conditions of the nerve tissue damaged in the hemisphere which permit the fits are analogous to various conditions seen by aid of the ophthalmoscope, at the ends of the optic nerves at the backs of the eyes. This positive sort of knowledge renders us less confident in our efforts at treatment, for when we have the optic discs under our eyes, showing acute changes, we feel keenly how little we are likely to be able to do. This, however, is the stage where treatment may, we trust, some time be useful. And the corresponding stage—I speculate—of the disease of the hemisphere which gives rise to or permits the unilateral seizures is the stage in

which we ought to interfere. Unfortunately, optic neuritis being frequently overlooked, we too often see only the permanent stage of optic neuritis, the stage of atrophy. And more often still we have to treat the permanent atrophy (?) of the nerve fibres of parts of the hemisphere which permits epileptiform seizures. It requires time for any sort of local damage to the cerebral hemisphere to bring about either the changes permitting epileptiform seizures or the changes which cause amaurosis, or, as in Case XX. &c., both.

In recapitulation, although this patient's (Case XX) "blindness and fits" probably depend as a *matter of fact* on syphilitic disease of the brain, as a *matter of idea* they depend on *coarse disease* of the brain. However convenient it may be to speak of cases like this, or say Case XIII., as "Syphilitic Diseases of the Nervous System," yet if we dispose of such individual medical problems by merely naming them thus, we shall not, I fear, think of our cases precisely, and treatment according to name will not prolong the satisfaction a superficial nosology first gives. These two *symptoms* are not syphilitic in any true sense. They will come on as the result of damage to the hemisphere of many sorts. Injury (see Case XVIII, p. 74) gives rise to just the same symptoms. I do not say it is not convenient to speak of a certain clinical group of cases as "Syphilitic affections of the Nervous System." Nor do I deny that syphilis may directly affect nervous tissue. However, I know nothing of the harm syphilis may do to the Nervous System, except as it acts by producing disease of the connective tissue in or about nervous organs and their blood-vessels. (See Note on Substitution Nutrition.)

Let us look at the subject from another point of view. There (Case XX) is a case in which there is or was hemiplegia from disease of the brain, and there is amaurosis too; or if the reader prefers not to speculate on the case of a patient who is still living, let us take Case XIII. Now,

before Helmholtz, we should have thought the amaurosis was, so to speak, syphilitic, and probably still those who never use the ophthalmoscope, would put down the defect of sight to syphilitic disease of the choroid. Similarly, now that it is known that defects of sight are sometimes due to retinal changes associated with those general changes which occur with chronic Bright's disease—when a patient in whom the general changes are marked is hemiplegic and blind—it is sometimes taken for granted that he has a clot in his brain, and a particular degeneration of retinal tissue, and perhaps clots in the eyes as well. This kind of reasoning is unsafe. It is true (see a case I have published, Lond. Hosp. Reps., vol. i, p. 384) that hemiplegia and blindness, each being the result of disease of a similar tissue—the choroid of the brain and the pia-mater of the eye—are sometimes found together. Again, I have (Lond. Hosp. Reps., vol. iii) published the case of a man who was hemiplegic from clot in the right optic thalamus, and blind from those retinal changes which sometimes occur with granular kidney. But, as a matter of fact,—now that we can see, if we will look, what kind of amaurosis really does occur with hemiplegia,—we find that the two events, the blindness and the paralysis, most frequently occur together,—not because similar or allied tissue changes have led to distinct damage of two organs, but because there is local coarse disease of the brain, from which coarse disease other changes diffuse. And the fact that that local disease is syphilitic (Case XIII.), tumour (Case II.), or blood clot (Case XI.), is not the point of most importance as regards the production of optic neuritis. The idea is the same, or similar, in each case; in each there is a "foreign body" in the brain.

So then the Tissue changes to which I first referred—disease of connective Tissue—are not *the* Tissue changes on which the epileptiform fits or amaurosis directly depend. The two sets of changes may be distinguished as Local

and Diffused.* Much of the hemisphere may be damaged without obvious symptoms of any kind, and it is doubtless only when the changes diffused around the damaged part reach the motor tract, or the convolutions near it, that the patient is liable to suffer from convulsions. In what direction the changes producing the accompanying amaurosis extend it is not easy even to speculate; but we must speculate before we begin our minute observations. And what the nature of these secondary changes may be is not yet properly known. Theoretically, I should imagine that lowered nutrition from any cause not leading to decided breaking up of the structure of the motor tract, would induce or permit convulsions of those muscular regions connected with it. If, however, the structure of the motor tract were actually damaged, there would be, if the damage were otherwise enough to produce any symptoms at all, one would think, not *disorder* of function—spasm, —but loss of function--paralysis.†

* These two expressions are clumsy. I do not like to use the terms Primary and Secondary, as I have used these expressions in a different sense. I have called changes in nervous tissue, beginning in nerve tissue itself, Primarily nervous, and those consecutive to disease of connective tissue, arteries, &c., Secondarily nervous.—Brit. Med. Jour., Nov. 24, 1866.

† I purposely avoid the use of the word softening in speaking of the changes about brain tumours. I may here say that I have no doubt that the whole of clinical evidence points to this conclusion which I draw from Dr. Radcliffe's researches, viz., that disordered function,—spasm, pain, incoherence, subjective sensations, &c.,—occur with enfeeblement, primary or secondary, of nerve tissue. The manner of production or of permission of convulsions from local disease of the hemisphere is a subject of extreme interest. And it is from those cases in which we can during life see something of the changes in part of a nervous organ, if it be only the end of a diseased nerve bundle, and not from cases of "genuine" epilepsy, that we shall, I think, begin to learn something of the nature of the permanent internal changes convulsions depend on. The contrast of *permanent* blindness and *occasional* fits is not the essential contrast. There are reasons to think that optic neuritis is the strict analogue of the *changes* producing or permitting unilateral epileptiform seizures. It may be, that the enfeebled hemisphere, or scattered parts of it, acquires a certain amount of tension from the nutritive changes of which it is capable, and that the tension is discharged through the *healthy* corpus striatum. Such speculations have, I think, this value, that they may

I now speak more in detail of the convulsive seizures. Amaurosis and these convulsive seizures must be studied together, if for no other reason than that they occur in connection with gross damage of the same part of the brain (see cases in last paper). I think it is certain that such convulsive seizures are the results of disease of some part of the hemisphere only, but the amaurosis, as I have several times urged, occurs with disease very variously seated; generally with obvious disease, as seen after death, in Case XVI, or as seen during life, Case XVIII. Although, in this paper, I speak of cases of convulsive seizures in which there is probably *obvious* disease of the hemisphere, I think it most likely that slighter changes, such as those resulting from embolism of the branches of the middle cerebral artery, will give rise to, or may permit, the same sort of convulsive attacks. Nay, in most cases of unilateral convulsions I should not expect to *discover* anything wrong in the Nervous System, although I should feel confident there was something abnormal in one cerebral hemisphere. In most cases of chronic unilateral convulsions there are no changes to be seen in the eyes. In a few there is irregularity of the retinal veins, and—to use terms—rudimentary neuritis.

Work at one of the symptoms will, however, help the work at the other; and I feel pretty confident that he who finds some of the steps of the process by which the changes of optic neuritis result from a foreign body in the cerebral hemisphere, will have learned much which will help research on the Laws of the slight, and yet widespread, Tissue changes, producing many obscure cerebral symptoms such as spasm, delirium, incoherence, &c. (See last footnote.)

The things most wanted are microscopical examinations

guide our microscopical inquiries to the exact position and nature of changes which occur in connection with disease of the hemisphere when unilateral epileptiform seizures and amaurosis, or unilateral paralysis and amaurosis, have been the symptoms during life.

to show on what structural changes of tissues diffused from "foreign bodies" the amaurosis and epileptiform seizures* depend. I hope Lockhart Clarke will tell us something of these changes in the parts I sent him from Case XXV.

To get to know in what region it is hopeful to look for the changes, we must do much clinical work before we begin our minute researches after death. In the specimen sent to Clarke the disease was so close to the optic thalamus that the Optic Nervous System may have been directly involved. The kind of cases which will help us to this knowledge are, I submit, those now considered in which there are symptoms of muscular disturbance of one side of the body, as unilateral palsy, or unilateral spasm, for these symptoms point, I feel sure, to a particular cerebral region, although to rather a wide one.

In the cases in which amaurosis occurs with epileptiform seizures (see especially Case XVI.), there is, as I have said, usually *coarse disease* of the hemisphere, and, in beginning to work at "epilepsy," I should begin—not with cases in which there is likely to be nothing discovered at post-mortem examinations—but at cases in which there is obviously something grossly wrong with the nervous

* I think it just as likely when symptoms like spasm of one hand or of one side of the body, or coloured vision, or a disagreeable smell come on, that the parts of the nervous system, on disordered function of which these phenomena depend, fail in some periodical change of the whole organism. An epileptic fit shews, I imagine, imperfect equilibration of a part with the whole. An emotional condition—perhaps we may say an arterial wave, *i. e.*, an affection of the whole heart of the body—sometimes gives rise, as we all know, to spasms of partially paralysed muscles. I do not neglect to consider, however, that enfeebled parts of the nervous system may fail because their own time, *i. e.*, the relation of nutrition to the possibilities of function, is not in harmony with the time of the whole organism. They have ceased to be physiological units. In Ague the periodicity, however much it may be manifested through the Nervous System, must, one would think, depend on alteration of nutritive changes, general or central. These questions are important ones for the consideration of ophthalmic surgeons as well as physicians.

system, as in Cases XVI. and XVIII. For the question is not what "causes epilepsy," but what conditions of altered nutrition and structure of nervous organs and tissues give rise to, or permit, disorder of function of any sort, viz., spasm of muscles, coloured vision, delirium, &c. From this point of view I earnestly beg that ophthalmologists will give epileptiform seizures a large share of their attention. And I ought here to remark more particularly that I do not say simply that Amaurosis in Case XX. occurs with Epilepsy. Not that this, according to a common use of terms, would be a wrong phrase, but because the term epilepsy has not a sufficiently precise meaning. Besides, this expression has not vitality in itself. It is extremely important to describe the sort of fit an amaurotic patient may have; and to call it "Epilepsy" gives the symptom too elastic a meaning, and careless people might think they had disposed of this part of a case by saying "the amaurosis is complicated with epilepsy." This classification would, I think, be an approach, under the cover of a conventional and yet an unreal preciseness to the analogous looseness, of saying "the amaurosis is complicated with paralysis," leaving it doubtful what sort of palsy there was. We want positive information as to how a convulsion is a *departure* from health of muscles and muscular groups and health of nervous organs and tissues, and not as to how far it *approaches* our idea of the almost metaphysical conception "genuine" Epilepsy. Whilst there are plenty of unsatisfactory "explanations" which cloud over our entire ignorance of the real seat and positive nature of tissue changes in the nervous system in most chronic convulsions, there are to be found on record scarcely any positive statements of what has really happened in particular convulsive paroxysms—a process which sometimes occurs under our very eyes. I am fully aware that there are admirable accounts of the worst fits as types, but some of these accounts are descriptions more of dramas of great

human interest than calm and cold scientific observations in an orderly sequence of the outward phenomena of an inwardly suffering nervous system. A tumour in the brain, at all events, is not likely to produce very exactly any form of "Epilepsy" described by the best authorities; and tumours, and other coarse forms of disease, are the usual intracranial changes which are associated with amaurosis.

Now, it is true that we rarely have the chance of getting a full and accurate account of the sort of fits our patients, amaurotic or not, have, but we can cautiously record what they and their friends tell us. It is especially important to get to know from them how the fit begins. But in a few cases there is no warning of the seizure, and no evidence to shew what physiological events really happen in any part of the paroxysm. Here we must be content to be ignorant and seek for better modes of investigation. It may then be allowable to say of a case that the amaurosis occurs with epilepsy, provided such an expression, whilst it implies that amaurosis occurs with general convulsions coming on periodically, implies also that a proper account of the paroxysm was not to be obtained.

Now, I have already suggested that unilateral convulsive seizures point to disease of the opposite side of the brain as unilateral paralysis nearly always does. I may say also that I feel convinced that unilateral chorea does too.* I do not mean that there is always disease which

* I have (Brit. Med. Journal, Sept. 22, 1866; Med. Times and Gazette, Dec. 1, pp. 584 and 585), suggested that unilateral chorea, unilateral convulsive seizures, and unilateral paralysis should be studied together, for facts towards ascertaining a knowledge of what I may here call the Laws of Muscular Life, and also for a knowledge of variations of muscular life in Time—both Time in the sense of the age of the patient, and in the sense of the time of the organism itself, or of the part injured. These three "diseases" are, I submit, due to variations of but one healthy state of the nervous matter of parts in the region of the middle cerebral artery. I may add that I distinguish those cases as unilateral in which the fits began in some muscles of the limbs on one side, or affect those on one side very much more than the

we can see without the microscope in cases of unilateral spasm, and everybody knows that there very rarely indeed is any, if ever, with unilateral irregular movements. Nor, it may be well to repeat, is unilateral paralysis or unilateral spasm often associated with amaurosis when the experience of ophthalmic surgeons and that of general physicians are put together. And the association, when it does happen, does not at present *seem* to be more than an accidental one.

Now, whilst every one grants that hemiplegia commonly points to disease of the higher parts of the motor and sensory tract,—the corpus striatum, and the optic thalamus,—I dare say few will admit that unilateral spasm usually points to disease in the very same region. No one is asked to accept any of these speculations, but this need not prevent the distinct study of cases in which defects of sight happen to occur with disordered states of muscles, be they what they may.—Constant Paralysis, Occasional Convulsions, or Continuous Irregular Movements, of a *certain Physiological Region*, *e.g.*, the muscles of the limbs. Such a kind of work will be more hopeful than working at cases of amaurosis as they are "complicated" by the entities "Epilepsy," "Paralysis," or "Chorea." There are,

other. In some attacks of this sort, the other side of the body may be convulsed after the convulsion in the one first affected is over. Unless a patient's friends are unusually intelligent, we get no information worth much as to the real nature of the full paroxysm. Then, I mean unilaterally as regards the limbs, but the trunk is also affected (see next footnote) in the complete paroxysm.

Until physicians work at the muscular disorders of various convulsive paroxysms as carefully as ophthalmic surgeons do at paralysis of the ocular muscles, our knowledge of convulsive disorders will not advance in an orderly way. The ophthalmologist would not tolerate the describing the phenomena of various visual illusions, produced by paralysis of muscles of the eye, as "Strabismus;" and we ought to try—although this is a far more difficult matter—to get faithful descriptions of actual occurrences in fits. And it must be admitted that it requires very high descriptive powers to describe what takes place in some cases of temporary disorders of motor function of the Nervous System.

very fortunately, cases in which the symptoms of unilateral muscular disorder refuse to be classed as either unilateral epileptiform seizures, or unilateral paralysis, or unilateral chorea. These cases are, I hold, of extreme value in leading to an organization of our knowledge of altered physiological conditions higher than an arrangement of symptoms in groups as epilepsy, chorea, &c. Ophthalmologists will be the last to purposely put amaurosis in association with the abstractions we call "genuine epilepsy," "real chorea," &c., when it is at all possible to avoid doing so.

I have suggested* that epileptiform seizures occur as the result of disturbance of the function, not only of many and of various divisions of the brain, but of parts in particular arterial regions, and that unilateral seizures are the counterparts of hemiplegia, such as results from plugging of the† middle

* As regards epilepsy, it would be better to think from the general law, if it be a law, that any region of the nervous system may *temporarily* fail in function. And, as I have said, instead of trying to see if in any one disorder the whole of the symptoms constitute "genuine" epilepsy—whether, for instance, real, true epilepsy is always attended by convulsions, or always "causes" loss of sensibility, &c.—I would describe disorder of function of the nervous system, or part of it, whatever it was, from spasm of the hand to general convulsions, from temporary coloured vision to spectral illusions, from occasional vertigo to actual incoherence. We might then, if the study of the phenomena disclosed any order with known healthy states, speculate on the organ, or organs affected, and on the Tissue changes which give rise to each of the disorders of function. I would, however, as I have said, *begin* the study of disorders of nervous functions by working at those disorders in which the outward phenomena are most simple and definite, *e.g.*, unilateral convulsions, in some of which the local disease is decided.

† When the convulsion is of the right side there is occasionally temporary defect of speech after the paroxysm, but this, I believe, occurs sometimes when the left side of the body is convulsed. This is a point to be studied. I used to call this defect Epileptic Aphemia (see Med. Times and Gazette, April 28, 1866). This temporary loss of speech may be compared and contrasted with temporary loss of sight—epileptiform amaurosis—which sometimes occurs with it.

In hemiplegia, from disease of the corpus striatum, the paralysis is chiefly of the limbs and not of the trunk ("Lectures on Hemiplegia," Lond. Hosp. Reports, Oct., 1865, vol. ii.) A little of the face is affected, and I

cerebral artery. These seizures occur, I speculate, as the result of impairment of nutrition of parts in the region of the middle cerebral artery, which impairment does not go on so far as to destroy the structural integrity of much of the motor tract. In cases where the epileptiform seizure is followed by hemiplegia, the structure of the motor tract must be itself degraded, more or less, as the degree and permanence of the paralysis shew. This speculation on arterial regions is an unverified one, and its only claim I urge is that

have (*op. cit.*) drawn attention to the fact that a hemiplegic patient may be able to close both eyes when he cannot close but the one on the paralysed side alone. Now in unilateral convulsive seizures (see Case XVI, p. 72) the trunk is convulsed too. Moreover, there was, in the case referred to, lateral deviation of the eyes to the side of the body convulsed. M. Prevost has, in a valuable paper, Gaz. Hebdom, Oct. 13, 1865, recently drawn attention to the fact that lateral deviation of the eyes, from the side paralysed, occurs for a short time in some cases after sudden cerebral hæmorrhages which produce hemiplegia. This symptom has, however, I learn, been described by Dr. Gull, in his Gulstonian Lectures, Med. Gazette, 1849. Now, it is, I submit, most important for ophthalmic surgeons to note the resemblances and differences betwixt hemiplegic palsy, and hemiplegic convulsion, if it be only because they will observe facts bearing on the physiology of the movements of the eye. Yet the study of one isolated symptom would lead through narrowness to incoherence. I referred to this symptom—speaking from the researches of Vulpian, Clarke, and Hutchinson—Lancet, March 24, 1866—chiefly with the object of shewing the importance of deviation of the eyes in epileptiform convulsion (see papers by Dr. Humphry, Mr. Lockhart Clarke—who long ago told me of his observation of this symptom in hemiplegia—Dr. Russell Reynolds, Dr. Broadbent in the Lancet, vol. i, 1866. Mr. Hutchinson has been for several years in the habit of pointing out this symptom as one of those which occasionally occur with laceration of the brain). I now wish to allude to Dr. Broadbent's researches more particularly—Lancet, May 5th, 1866—on this symptom. His views seem to me to put this odd and curious temporary muscular disorder in its proper place with other symptoms in hemiplegia. The phenomena of some unilateral epileptiform seizures, *e. g.*, Case XVI., p. 72, confirm, I think, the views he has put forward as to how it is that the muscles of the trunk escape in the common form of hemiplegia, and his views help very much to make clearer the hypothesis I have put forward as to the relations of unilateral chorea, unilateral epileptiform seizures, and unilateral paralysis. In cerebral hæmorrhage the eyes are turned from the paralysed side in the direction in which the face is deviated. In Case XVI, in the fits I witnessed, the eyes were turned, as the mouth was drawn, to the side of the body convulsed.

it seems to lead to a reasonable method of investigating the order of symptoms in disorder of function of the brain. If it leads to work on a methodical plan it will do good. But if it check work by "explaining" things, it may be suspected of want of that inherent vitality which true method has in itself to open out more and more work.

But in Case XX. there was, *i.e.*, in the first fit, besides the unilateral muscular symptoms, disordered vision; and in plugging of the middle cerebral artery, vision is not even temporarily affected, *so far as I know.* I will now speak of the temporary defect of sight, and then return to this discrepancy.

This, at that time, trivial symptom to the patient, had to me great interest. As the unilateral convulsion was followed by unilateral paralysis, so the temporary failure of function of the eye* was followed by loss of its function.

More frequently, I believe, speaking of unilateral epileptiform seizures, just the reverse happens; the temporary disorder of sight rarely passes into a loss of sight; whilst the temporary disorder of the muscles of the limbs of one side not unfrequently passes into paralysis. What connection there may have been betwixt the "eye symptoms" and the "side symptoms," it is impossible to be sure, but it is fair to speculate. I have suggested, Medical Times and Gazette, April 30, 1864, that they were connected in this case, because the middle cerebral artery supplies parts of *each* optic nerve (*i.e.*, including commissure and optic tract), as well as the corpus striatum, and much of the hemisphere, and that the whole of the parts in the range of this vessel suffered first all temporarily, and then all permanently. Here, however, the paralysis of the limbs passed off, so that the condition of the nerve tissue of the corpus striatum, *i.e.*, the corpus striatum as a whole, returned nearly to a state of health, whilst the optic nerves remained diseased. And no doubt there is some permanently abnormal

* I use the word eye, because I do not just now venture to speculate as to the exact part of the Optic Nervous System affected.

condition of the right hemisphere, which permits the occasional spasms of the left side of the face, left hand, &c.

I think the study of the divisions of the nervous system, according to its blood supply, is worthy the attention of ophthalmologists. Indeed, I feel pretty confident that work on this plan will enable us to arrange in a real order symptoms oddly mixed as disorders of function of parts of the Nervous System, especially those disorders that are temporary. And it seems to me to be reasonable to suppose that the calling out of healthy function of nervous organs, as well as disordered function, depends, *i.e.*, as one link, on alterations in blood supply; and that thus the arteries of the brain are not to be thought of altogether as pipes conveying nutritive material, and carrying away the excreta of organs, but as schemes also for the orderly development of possibilities of function. They have contractile walls and nerves. Whether the function of parts of the brain be brought out in health by actual contraction of vessels, as I have just suggested, or not, it is impossible to be certain, but the function of the retina is roused, in a disorderly manner it is true, by a pressure on the globe, which pressure tends to empty the vessels which supply the nerve expansion with blood.

Again, the disordered muscular action of the hand, &c., "partial fits," which now and then comes on in patients subject to complete unilateral spasm, as in Cases XVIII. and XX., and which, indeed, often precede the full attack, seems to me to be undoubtedly due to feebleness of nerve tissue, as the muscles occasionally convulsed are often permanently, although partially paralysed. The blood binds together the vegetal life of all tissues and the functions of every organ. And the arteries of the brain evidently knit up scattered threads of function. Here the warp of Life is seen distinct from the inseparable woof of Function. Parts which have no relation by continuity of function have one by community of nutrition, and thus suffer together in disease. Hence it happens that we find symp-

toms so oddly assorted as subjective sensations of smell, spasm of muscles on one side of the body, insensibility, and when the whole fit is over, defect of speech (see Medical Times and Gazette, 1864, vol. i., p. 168), or as in Case XX., subjective visual symptoms, spasm of one side of the body, and insensibility.

The above speculation is, as I have already said, an unverified one, but it may, I think, give order, at least for a time, to some of our scientific labours. The arterial relations of the whole Optic Nervous System ought to be carefully studied for the most common reasons.

I some time ago tried to show—although I succeeded very imperfectly—that the parts of the Nervous, Muscular, &c., Systems might be arranged on a plan corresponding to that of the Vertebral Scheme of Professor Owen. I still think there is much truth in this idea. The line of order in such a scheme is one according to continuous differentiations of functions; and, I imagine that the order of another scheme may be from development according to lines of nutrition or of arterial supply.

As Case XII. shews (see also footnote, p. 436), damage quite limited to a part of the hemisphere sometimes gives rise, at the exact time when it happens, to coloured vision. It would be easy to say, adopting some of the views of the day, that the sudden tearing up of structure caused a liberation of tension, which induced contraction of the vessels of the whole arterial region, including the optic nerve, in which the part damaged lay; or that the symptoms arose from "ground currents" affecting wide-spread fibres of the optic nerves, and also those going to the limbs. Indeed these views may each contain an indistinct view of the truth. I have several times alluded to the fact, that disease of one of the hemispheres gives rise to amaurosis, and to the fact that apparently either of them may be diseased without any defect of sight. This last remark applies equally to epileptiform seizures, and, probably, to the intimate changes

which permit them. As I suppose each hemisphere receives fibres directly or indirectly from each retina, "irritation" in *one* hemisphere might produce *temporary* affection of sight in *both* eyes, although local damage might not produce *permanent* defect of vision.

We shall, I believe, receive hints as to the explanation of the occurrence of optic neuritis of both eyes from disease of one hemisphere, by considering from another point of view the relations of unilateral spasm and unilateral paralysis. (see footnote on Deviation of Eyeballs.)

In hemiplegia the muscles of the trunk are not paralysed when the corpus striatum is damaged. Now Dr. Broadbent* accounts for the escape of the muscles acting bilaterally by supposing—not that these muscles are unconnected by fibres with the higher parts of the motor tract—but that, through a common nucleus, they are doubly connected; and, thus, when one corpus striatum is damaged its fellow is still sufficiently connected with the common nucleus to empower the muscles acting together on the two sides. Now, it seems to me that the phenomena of Case XVI., in which the corpus striatum was, so to speak, "stimulated," confirm Dr. Broadbent's hypothesis. In that case, the muscles of the trunk were in spasm, because their nucleus was "stimulated" from *one* corpus striatum. Dr. Broadbent, I understand, applies his hypothesis to sensory as well as to motor phenomena. The fact that disease of *one* hemisphere is attended by amaurosis of *both* eyes may seem to negative the idea that each hemisphere receives fibres representing each eye. But we must remember that disease of even a very large part of the hemisphere *does not produce* amaurosis, although —I may seem to be speaking paradoxically—amaurosis not unfrequently occurs as a consequence of damage of one hemisphere. Amaurosis from optic neuritis is not due

* See Dr. Broadbent's paper, Med. Chir. Rev., April, 1866. An abstract of it is published in Ranking's Abstract, vol. i., 1866. Lancet, May 5.

to a loss of *function* of some nervous structure, as loss of speech or hemiplegia is, but is evidently a part of wide nutritive changes, which spread, it is most probable, not along nerve fibres, but along the connective tissue of blood-vessels, in arterial regions—possibly in vaso-motor regions. The phenomena of unilateral epileptiform seizures at least illustrate the production of double amaurosis from disease of one hemisphere.

Yet, in spite of the facts which appear to show that the position of disease, so far as producing optic neuritis goes, is of little consequence, I do not attach so much value to any of my speculations, but that I would still urge a more careful examination to render this more certain, or to show that it is only partly the truth. I am most anxious to keep the annoying difficulties of the subject before me. I have no wish to conclude, and the speculations I put forward are only of value to me so far as they stimulate work and give order to it. Independently of any theory, we ought, I submit, to locate the damage to the hemisphere, which occurs with optic neuritis, as carefully as we can, especially when the disease is near either of the corpora striata. Although widely distributed, the optic nerve fibres may yet have particular foci, and it may be that these foci are situated so far forward as, or even anterior to, the corpus striatum. Whatever hypothesis might seem to be true, one would not deny that the nerve fibres of each of the special senses, even those of hearing, some more and some less, spread directly or commissurally to every part (although more perhaps to some parts) of their periphery, viz., the convolutions of the cerebral hemispheres. There seems to be reason for believing that every part of the arm is represented in each part of the corpus striatum, and that, thus, the corpus striatum is, in Spencer's sense, a mass of physiological units. Similarly it may be that the special senses, &c., are represented widely in each cerebral hemisphere, and thus that much even of both the hemispheres may be

destroyed without affection of sight. I may just remark that I believe the complexities of nerve fibres and cells are best studied from a consideration of the comparatively simple experiments damage of the corpus striatum makes. To this part of the question of Evolution of Movement I have referred in a footnote to a Lecture on Cerebral Hæmorrhage, London Hospital Reports, vol. iii., p. 238-9.

Indeed I confess that, in the absence of more precise facts, speculations on the relations of coloured vision and spasm of one side of the body, are not very profitable. We want first, I am convinced, more observations on the gross associations of the symptoms, in many cases, one to another, before we can speculate on their causes, or why they come together; for coloured vision leading to amaurosis, and unilateral muscular disorders are not, as I have said, often found together. Complete amaurosis with either hemiplegia or unilateral spasm is not common.

It may be urged that temporary defects of sight are unimportant. Perhaps they very often are in a narrow practical sense. In the case I have just related such a defect of vision was a warning of real evil. This, however, was not a simple case of epileptiform amaurosis. But I willingly confess that I do not know what ill meaning temporary defects of sight have, as I have not yet been able to trace many cases. It is important to consider Case XII. in which temporary coloured vision, from tearing of a particular part of the brain by blood-clot, was followed some time after by amaurosis.

Such symptoms may, I think, be usefully put in the same category for study—not put aside with them—as the so-called auræ of epilepsy or partial fits. If a man were to have twitchings of one hand, of one leg, or of one side of the face, I should fear, although I should not be able to predict with a feeling of certainty, that he would have epileptiform seizures of much wider range, and that he might have unilateral paralysis. A case of this kind I have recorded—*Medical Times and Gazette*, January 31,

1863. So, if a patient were to have sudden and temporary coloured vision, or temporary loss of sight, I should fear such a symptom might be the first warning of severe cerebral disease, in which amaurosis would be one of the symptoms. No doubt, attacks of temporary loss or defect of sight may occur for years without any very bad permanent results. The sight of the woman Julia W., whose case I recorded in a previous paper, has remained good. Still I know that complete unilateral convulsion may occur and not be followed by paralysis for years, and hemiplegia may then come on. And, in the same way, I should fear that temporary disturbance of the functions of the retinæ might depend on slight changes in the brain, on increase of which, loss of visual function might follow.

I was once consulted by a cabman whose sight left him temporarily altogether when he was driving. I could not ascertain from him that he had had any other symptoms. Some time afterwards he told me that he had learned that his wife had kept from him the fact that one night he had a convulsive attack, before he consulted me for temporary defect of sight. In this case the temporary loss of sight was doubtless part of a fit—an abortive fit.

I have considered together temporary loss of sight and temporary coloured vision. The former seems to be the analogue of loss of sensation and paralysis; the latter of pain and spasm. As I have said, I adopt the views of my colleague, Dr. Radcliffe, so far as this, that I believe pain and spasm are signs of enfeeblement, and not of exaltation of nervous function.

I may, before leaving this part of the subject, mention that Mr. Zachariah Laurence has written a very able and interesting paper in the British Medical Journal on functional affections of the retina.*

* These subjective sensations, properly considered, lead us to think of the vast modifications of sensation which occur in the hemispheres, as well as of the modifications of motion—of various degrees of Perception, as well as of various degrees of Expression. And just as—to take a limited part of Ex-

Such symptoms are often called Functional. If Functional means that the alteration of nutrition is so slight that health is apparently easily and perfectly recoverable, well and good; but if it mean that the symptom is not of serious import, I should question the advisability of using the term. I fully admit, then, that I do not know the meaning temporary coloured vision has to the doctor, and I do not think it profitable to speculate on its cause, except so far as the speculation opens out practicable fields of research. I have no doubt that temporary and definitely recurring attacks of coloured vision do depend on alterations of tissue in some part of the nervous system, just as I have no doubt that temporary and definitely recurring spasms of the hand do. Our work should begin

pression—we think of degrees of all defects of motion from the imperfect utterance of words up to incoherence, so we should think—to take as an illustration a limited part of Perception—of all defects of sensation from coloured vision to spectral illusions. Defects of speech occur from damage near the corpus striatum, because here the groupings of motor or motor-impulse nerves narrow towards a connection with the immediate instruments of motion. This knowledge of location of damage which disorders the most elaborate form of expression by motion—I do not say the seat of some conventional bundle of motions tied tightly by technical words into a "faculty"—will urge us to ask where the complex impressions from the several sense systems—not "end" but—embrace the cerebral systems of motions and motor-impulses. It is easy to say this point is the thalamus opticus. Indeed it may be that here the various threads begin their simplest interweavings, as the groupings of fibres for the simplest weavings of muscle in higher mental operations seem to be near the corpus striatum. But I should think the highest groupings of sensory nerves are continuous, deep in the cerebrum with the highest groupings of motion.

Generally I have taken defects of motion as illustrations of a possible common ground of work betwixt ophthalmologists and general physicians. It is obvious that motion and sensation are everywhere interweaved. In the eye sensation is chief; motion subordinate; in articulation sensation is subordinate and motion chief. In one motion is the servant of sensation; in the other sensation is the servant of motion. In the higher evolutions of motion and sensation, no doubt, the two phases of nerve action are so intermingled that incoherence (whatever the most striking phenomenon may be) is a defect in the highest evolutions of both motion and sensation, of their widest interrelations and associations.

by studying phenomena, and part of it should be, at least, I think, an attempt to define most carefully the circumstances under which these sudden failures occur, and we may hope then to get to know on what they depend and what they foretell. In my experience, when these temporary defects of vision are accompanied by any local symptoms, the most frequent one is numbness or spasm of one side of the body. However, in severer epileptiform seizures, the defect of sight will be, as it were, covered up by general insensibility.

Now, I confess that we are likely to get wrong by studying things very minutely, as well as by studying cases broadly. I recognize also the danger we are in in receiving the reports patients give of their subjective sensations. In spite of these drawbacks, as such symptoms exist, they must be studied as well as we can study them, and we should attempt to put them in some reasonable kind of *order*. Temporary loss of sight occurs alone, and I have called it Epilepsy of the Retina. This term perhaps is objectionable in that it carries the theory that the retina is solely at fault. Epileptiform amaurosis might be less objectionable. Such terms are useful so far only as they put these temporary defects in the right group for study, but worse than useless if they put an end to investigation by "explaining" them away. The affinities of these temporary failures of function of the optic nervous system are, I really think, undoubtedly with chronic convulsive seizures, with temporary loss of speech, and with temporary states like somnambulism. These epilepsies must all be studied, in order that by arriving at the knowledge of the things in common we may get a natural plan of studying each individual epilepsy as a departure from health of Tissues, Organs, and Functions of the Brain. The ophthalmologist has excellent opportunities of studying temporary disorders of Function of the Nervous Tissues of one Organ, viz., the retina. Temporary defect of sight is, I think, the same in *idea* as is the tem-

porary somnambulism into which some people pass as suddenly as others lose their sight or their power of talking. They are both temporary conditions of tissues of nervous organs, although of very different organs—the retina (?) and the hemisphere.

Here Case XXI.

This case is one of great importance. Looked at superficially, it resembles Case VIII. The subjective sensations of smell, sight, and hearing, which this patient had, are extremely interesting; and these, considered with the spasm of one arm, the subjective motion, shew that we have to consider variations of the health of the optic nervous system in relation with variations of the healthy states of many other parts of the nervous system, and not as to whether epilepsy "causes" amaurosis, &c., &c. To speculate on the internal conditions on which the whole of this patient's symptoms depend would not be very profitable. It would not be difficult to bring them into order according to the views of the day. The difficulty of walking might lead some to think the disease is of the cerebellum.*

* In general, disease of the cerebellum is the last thing I think of when I see a patient walking in a disorderly manner. In fact, disease of the cerebellum, *i.e.*, disease of the cerebellum so limited as to constitute a physiological experiment, is a very rare thing indeed. Abscess of this part of the Nervous System is so attended by acute general symptoms that cases of this kind would not often be proper evidence. Then disease is often limited to one side. I would here, however, urge, as one fragment of study, a general consideration of the cases in which optic neuritis occurs with difficulty in walking, *i.e.*, a difficulty without obvious paralysis. Such cases should be compared and contrasted with cases of Locomotor ataxy. I have now under my care a case (see report, Lancet, March 31, 1866), of Locomotor ataxy, in which the phenomena, superficially read, are like those of Betsy S.'s case. I have remarked on the class of cases to which Betsy S.'s belongs, Med. Times and Gazette, June 17, 1865. For fear of being misunderstood, I may say that I only group optic neuritis with difficulty of walking as a mere approximation to studying certain phenomena. The sort of difficulty of walking must be studied in each case, and in short we want, as I have urged in several places, a knowledge of the laws of movement from health to paralysis, rather than accounts of cases of Locomotor ataxy, &c.

The cerebellum may be involved, but I think the disease cannot be limited to one of its lobes, or there would not be so much difficulty of movement. I think the disease must be chiefly of the left cerebral hemisphere, and probably of its anterior lobe; but I equally think there must be disease lower down, and away from the cerebral hemisphere, to produce the deafness. For, so far as I know, deafness to any considerable extent does not occur from disease of the cerebral hemisphere. This may be due in part to the wide connection which hearing probably has with mental functions. Probably fibres from the auditory nerve through its nucleus spread more uniformly to the cerebral convolutions. There can, I suppose, be little doubt that the auditory nerve does send fibres to the hemispheres directly or commissurally. The chief reason of the escape of this sense, or of its being less affected, is, possibly, that its vascular (nutritive ?) relations with the hemisphere are less.*

The fact that a large part of the auditory nerve goes to the cerebellum might seem to countenance the idea that in this patient the cerebellum is diseased. The disease, if in the hemisphere, cannot be, one would think, very near the

* I have (Lancet, June 16, 1866), spoken of the importance of considering the relations of each of the special sense systems to the hemisphere [and of course to other parts] in three ways: 1st, Geographical; 2nd, Physiological (Functional); 3rd, Vascular (Nutritive). The olfactory and the auditory nervous systems stand in good contrast. (1.) The olfactory bulbs are near an important part of the great intellectual centre—the hemisphere. The auditory nucleus is continuous (Lockhart Clarke) with the vagus nucleus, the centre so-called of animal life. (2.) The functional connections of smell and hearing with the hemisphere, although not equally important, are equally significant. (3.) The vascular connections are widely different. The vascular connections of the olfactory nervous system ought, I think, to be very carefully considered. Subjective sensations of smell ought to be compared and contrasted with subjective sensations of vision. My experience of subjective sensations of smell and sight is too narrow to enable me to help much in this comparison and contrast. Alienists see the former, and Ophthalmologists the latter. I refer to one case (Case XXIII.). The geographical, functional, and nutritive relations of the whole of the sense nervous systems are worthy of the most careful study by Physicians, Ophthalmologists, Physiologists, and Psychologists.

corpus striatum, or there would probably be both some defect of speech and a little paralysis of the limbs of the opposite side of the body. Yet the unilateral movements in the fit the girl had, satisfy me that there is disease more or less nearly related to the higher part of the motor tract. I used the expression *in* the hemisphere, as I have seen a tumour which had grown in the fissure of Sylvius, and it had displaced the frontal convolutions; but not having involved them, nor the corpus striatum, there had not been either defect of speech or paralysis of the limbs of the opposite side of the body until the day before the patient's death. He had been amaurotic. The case was that of a young gentleman who had been under the care of my colleague Dr. Radcliffe and Dr. Ruttledge. In Betsy S.'s case it may be that a tumour has had such relations in the fissure of Sylvius. I imagine the loss of smell to be the result of disease in the hemisphere, and not to be caused by disease involving any known part of the olfactory nervous system. Whilst we know that disease of the hemisphere is attended by loss of smell, the way in which the symptom is produced by disease so placed must remain unknown. The speculation that anosmia occurring with cerebral tumours is often the result of an olfactory neuritis, may be a useful one for arranging our thoughts, and for guiding investigation, but it must remain a speculation for some time to come.

See Case XXII.

It is most important to give a place in our thoughts to the slightest one-sided symptom. Unilateral epileptiform fits may affect but a limited part of that physiological region which they occasionally fully involve. Thus there may be spasm of the face, of the hand, of the leg, and these symptoms are sometimes called by the name aura.* "First symptom"

* Here, again, I will urge the general study of varieties of movement to which I have so often referred. Taking one physiological region—that of the limbs—I would study conditions of muscles according to the part of this

is a better term, as it involves no theory, and yet gives us hints as to the desirability of studying what functional region or part of the brain is first disordered, in order that we may seek in a reasonable manner where the epileptic process begins in the brain, and thus get a clear idea of where to look, after death, for those minute abnormal changes which are the permanent part of that unknown process. In this way coloured vision and subjective sensations of smell, and limited subjective motions, are important, and with other symptoms may point to disease of particular regions of the brain; but we know too little of permanent defects of smell and sight to enable us to begin to conclude on the causes of temporary defects of these senses.

It is quite clear that impairment of smell, from slight defect of the olfactory nerves, cannot be estimated as slight defects of the optic nerves can. Although this patient can smell peppermint, she may have a condition of the olfactory nerve as abnormal as the condition of the optic nerve in Case XXVI. That man thinks he sees well, although

region most affected, according to their *variations in time* from the voluntary movements of health, through nearly constant irregular movements, continuous rigidity, contractures, and occasional spasm to more exact periodical seizures. And I would, almost advisedly, be careless of naming cases from most marked set of symptoms, chorea, or epilepsy, or paralysis. The laws of the evolution of movements will, I think, be studied at the same time. When the spasm, of the hand, "partial fits" are witnessed, it will be found that they are not of one anatomical set of muscles, as of extensors or flexors, but that there is one contending spasm of all the muscles which go to the hand. It is a defect in the highest range of Evolution. The study of hemiplegia leads me to the belief that the whole of the arm is represented in each part of the corpus striatum (Lond. Hosp. Reps., vol. iii, p. 238), and I think the phenomena of unilateral chorea and of some unilateral epileptiform seizures also illustrate this. I imagine that, however much the hand may appear to suffer alone, it suffers in the most highly developed and the part most represented of the *whole* limb. In general every act is a physiological unit in Spencer's sense of the term. However, I am here supposing, what probably most of my readers will not grant, that unilateral palsy, unilateral spasm, and unilateral irregular movements are various states of defects due to changes in what I may call the middle-cerebral-artery system.

there are striking changes to be seen in his optic discs, and in his case, too, the olfactory nerve may be affected, although he can smell peppermint and taste cinnamon. It is indeed very important to bear in mind the possibility that, as is the case with the optic nerves, the other special sense nerves may be obviously damaged without great interference with their function.

The fact that there was no permanent or long-continued loss of speech in Mrs. S.'s case makes me think the disease cannot have actually involved what I may call Broca's region. The temporary loss of speech was due probably to temporary conditions of nerve tissue of this part—probably some may say to spasm of its vessels—changes which we believe to exist in epileptic hemiplegia, but which we have never defined. I have little doubt, however, that there is gross disease in the left hemisphere in some relation to the motor tract, if not to Broca's aspect of it.

Now this case is another of several exceptions to what I once thought to be almost a rule, viz., that disease of the right hemisphere is more likely to give rise to amaurosis than disease of the left one. (See Postscript, p. 306.) I stated correctly the facts I had observed. I was to some extent wrong in not looking for amaurosis in certain cases of right hemiplegia, namely, in such cases as those in which there was considerable loss of speech. See Cases XII. and XIV. However, I may excuse myself by a consideration of the fact that, as far as I know, no one has explored the eyes of speechless patients as a matter of routine. I have only omitted to do what every one else has omitted to do. The following case shews the necessity of using the ophthalmoscope in cases of loss of speech. In relating it I purposely avoid exaggerating the ophthalmological aspect of it. Indeed that part of the case is the least striking. I wish to give place to the whole of the symptoms with which amaurosis does occur.

Case XXIII.—(Richard A.)

Now in this case the patient's sight was not affected *because* a part of his hemisphere was injured. There can be little doubt that the corpus striatum was much broken up, and that many convolutions were damaged at the very least in the sense that their connections with the motor tract were to a great extent destroyed.* In the Mirror of the Lancet, Dec. 1, 1866, is the report of the case of a patient who was under my care in the London Hospital, and whose case was so like that of Richard A. that I think it most possible that Richard A. had a similar kind of damage to his brain. However, as there was no autopsy in Richard A.'s case, Case XII. may be compared with the case published in the Lancet. Now, in the latter patient there had been no changes observed in the optic discs up to his death.† This shews that damage to a certain important functional region of the brain does not produce optic neuritis, but is the indirect cause of it. I have considered—I by no means say I have explained—this apparent paradox in a previous page. It may be that, had the patient whose case is reported in the Lancet lived, he might have had optic neuritis, not because he had loss of speech, but because he had a foreign body in his brain. The fact that this foreign body was in this particular region was not of great importance so far as the production of amaurosis is concerned. I mean that the fact that it lay in a locality of high function does not appear to be a very important one. But as this region is also the most vascular part of the brain, the presence of a mass of disease here may be more likely to be followed by amaurosis than disease in other parts of the brain. Optic neuritis is not, however, due to an absence of a greater or smaller quantity of nervous

* The other day I made a post-mortem examination, in which there was a large clot outside the corpus striatum. There is in an uninjured brain no room for a large clot "outside" the corpus striatum. In this case the convulsions seemed, as it were, *split* from the corpus striatum and the clot lay in the gap.

† A fact which unfortunately I omitted to state to the reporter of the Lancet.

structure, as loss of speech is, but to the presence of a mass out of its place. The other day I made, with Dr. Hooper May, of Tottenham, a *post mortem* on the body of a man who had been speechless for nine years, and hemiplegic of the right side. Yet, although many convolutions of this man's brain seem* to have disappeared, there was no discoverable defect in his optic discs when I looked at them a few days before he died.

Yet, as I have repeatedly said, some parts of the brain are rarely diseased; for instance, I cannot call to mind that I ever saw a blood clot limited to one posterior cerebral lobe. Such points are of importance in our study of the supposed location of "faculties," as well as in our investigation as to the seat of damage which may or does permit amaurosis. For very many reasons the studies of ophthalmologists and physicians must be merged; and they should, I submit, begin, as one point, with amaurosis and loss of speech. Loss of speech is a defect of function. Amaurosis is a disorder of nutrition. The separation of function and nutrition is in one sense arbitrary, but it is at least convenient to look at certain morbid processes from different stand-points.

So far I have spoken of cases of defects of sight in which the changes in the eyes were well marked. But of course I pay attention to slighter changes. The greatest care, however, is necessary that we do not in this, as in other things, attach too much importance to slight circumstances. Again, I have heretofore spoken chiefly of those cases in which the amaurosis has depended on coarse intracranial disease. Indeed I think—I have not concluded—that slight, or wide-spread changes, *e.g.* those produced by embolism, are not often associated with optic neuritis. I trust, if I can decide this point, that the absence or presence

* I have not yet dissected this brain; it was soft and is in strong spirit to harden. This accounts for the general expression in the text. I have to thank my friend, Dr. Mackenzie Bacon, late of Fulbourn, now of Brighton, for this important case.

of defects in the optic discs will sometimes help us in our diagnosis of the nature of intra-cranial disease. From this point of view the examination of the eyes of patients the subjects of chorea is of importance. Yet I do this sort of work apart, so far as I am able, from any preconceived notions as to the state of the nervous system in chorea. At all events most persons are agreed that the changes in the Nervous System in this disease are slight. Indeed, so slight are they that physicians are not agreed, even roughly, as to their position. It is more a faith that there are changes somewhere, than a belief that there are discoverable changes anywhere. Of course the proper thing for us to do is to study first the phenomena in each case carefully, from the broad basis of the evolution of movement, and the conditions of nerve tissue anywhere permitting any kind of defect of movement, and from other general bases. Irregular movements of a sort do occur with spinal disease, but irregular movements in young children are usually different to those observed in spinal disease (as, for instance, of the arms of patients who have disease of the posterior columns of the cord), and point I think—I judge from clinical evidence—to disease of the cerebrum. These questions are really of interest to ophthalmologists, for although the first question may well be the relation of certain intra-ocular changes, when there are any, to particular irregular movements, such a question is only the prelude to wider considerations of what sort of changes of parts of the Nervous System cause or permit both the defect of sight and the irregular movements.

The Ophthalmological Study of Chorea.

The absence of decided defects in the optic discs in cases of chorea in children is a fact worthy the careful attention of ophthalmologists. I look at the eyes of children the subjects of chorea as a matter of routine, just as I examine their hearts as a matter of routine.

On September 6, I looked at the optic discs of Caroline B., a girl ten years of age, and, as I was sitting down, I remarked to the students that I had never found any striking changes in the eyes of a choreal patient. I found them in this case. The changes were most in the right eye. The optic disc was badly margined; the veins were large and very irregular (wavy); and the disc was hyperæmic. There were similar, but slighter, appearances in the left eye. Now, in this case, as in most cases of chorea, one side of the body—the right—was affected. It was the side only affected. There was, as I believe there always is in chorea, paralysis, and in this case the paralysis was considerable. The child's talking was bad. It was, when I saw her, slightly thick. I took her into the hospital (thanks to the permission of my senior colleague, Dr. Davies). The irregular movements degenerated, into paralysis, so that the arm was almost immoveable and the changes in the optic disc increased. However, she ultimately regained much power, and the discs resumed nearly a normal appearance. She went to a Convalescent Institution in November. The arm was then nearly restored.*

The treatment, the drug treatment at least, of chorea is, of course, interesting to ophthalmologists, as we suppose we are treating conditions of tissues, and not the phenomena, although we find our remedies empirically. Of drugs, Arsenic seems to be by far the best. I would investigate its action, not, of course, as a remedy for chorea, but as a remedy for impaired nerve tissue. Its

* I am pretty confident that this patient had had no severe pain in her head, and no vomiting; but, strange to say, I have no note of enquiries about these symptoms. It is very important to enquire on these points in cases of nervous diseases, as pain in the head and sickness are valuable signs to help us to distinguish whether disease in the head is coarse and local or slight and wide-spread. And it is of importance to make enquiries about these symptoms in all cases of chorea as well as to examine the eyes with the ophthalmoscope, and to examine the heart.

chemical relations to phosphorus—so important a constituent of nerve tissue—are very significant.

I may just mention the treatment of this patient's case. I think few nervous diseases require more care in treatment than chorea. I gave the child good diet, directed her to be kept very quiet mentally and physically, and yet to take exercise. No drugs were given beyond aperients; for, as Wilks has shown many, probably most, cases of chorea in young girls will get well by treatment without drugs. I think it of extreme importance to keep choreal patients quiet when the disease is in its *earliest* stage. I would disturb them in no way, even avoiding cold douche, and, in moderation, decided moral restraint, and, in short, anything that would frighten or excite the children. When the disease became at all chronic would be the time for gymnastics, or for more decided measures.

I use in the above report the vague expression that the child's talking was bad because I have not been able to do much towards analyzing the defective articulation of patients the subjects of chorea. This matter has, from a broad basis, a decided and a direct interest to ophthalmic surgeons.

When I was clinical assistant at Moorfields I used to observe that some patients waiting their turn were obviously squinting, but when they came to speak to me their eyes were apparently parallel. So choreal patients who are reported to talk very badly at home, can often pronounce any word I suggest. So it often is with a certain defective articulation which occurs with hemiplegia, generally of the right side. I have known a hemiplegic patient who talked so badly that it required careful attention to gather and guess what he said, and who could, for all that, utter any single word or sound I uttered. I repeat, these questions are of interest to ophthalmologists when they are thinking, *from* those striking muscular disorders they most often see, to the Laws of Muscular Life. The difficulty of articulation with hemiplegia (See

Case XIV.) is not to be analysed into difficulty in pronouncing palatals, as in diphtherial paralysis, nor dentals, nor labials, for there is no paralysis of any corresponding divisions of the articulatory organs. It is a defect in a higher range of evolution—defect in a higher series of anatomical possibilities.*

Note on Substitution Nutrition.

I may here allude to the fact that bromide of potassium is better than the iodide in cases of epilepsy, at all events in chronic cases. It may be well to give iodide with it to stop any new changes, but, as a matter of empirical experience, the bromide keeps off the fits far better. The treatment of the patient, Case XX., is, except when there is a new style of symptoms, just like that of Case XVIII., in which the gross local disease is the result of injury. As I have urged, the unknown changes on which the fits depend are in both cases probably similar, and, no doubt, quite alike in this, that the nutrition of certain tracts of the hemisphere is below par.

There can be no question that bromide of potassium is a most important remedy for keeping off fits—I do not say for curing the patient. It is, of course, not precise to

* On the evolution of movements I have spoken several times, especially as regards the arm-nervous-system, *i.e.*, from nerve-trunks supplying muscles directly, to the corpus striatum. From this centre we shall, I hope, be able to trace another series of evolutions of movements, those of speech and thought. I think, too, as I have already said, that this kind of study will be much helped by a consideration of what ophthalmologists have done in their work at paralysis of the muscles of the eye. And ophthalmologists will do more than study such phenomena as paralysis of muscles from disease of an anatomical nerve-trunk, just as physicians do more than study such difficulties of articulation as paralysis of the palate gives rise to. Ophthalmologists will observe carefully the relations and meanings of oscillations of the eyeballs and of lateral deviations of the eyes, which symptoms—the last, certainly—are most likely always caused by deep central disease. If such an expression be permitted, there is a gradual increase in *intelligence* in movements from the lowest nerve-trunks to the highest nervous centres. (See postscript, No. 2, p. 306.)

say that bromide of potassium is a remedy for Epilepsy, but for certain states of nervous matter. I use the word matter instead of tissue as it cannot be certain what is the particular tissue change the bromide helps in. Probably change in nervous tissue is the one it assists. At all events I would urge its use both in cases of the nerve-atrophy of the optic nerve (if I may be so tautological), and in acute optic neuritis, and in the cerebral-atrophy of the optic nerve which follows this acute process, and which atrophy seems to be only secondarily nervous. It ought, I think, to be tried in paralysis of the motor nerves of the eye, and generally in altered conditions of nerve matter.

How substances practically foreign to the organism act in disease is an important problem, and we should investigate their action more generally from the part salts, minerals, &c., play in healthy changes of nutrition, in building structure and in developing function. The chlorides, the iodides, and the bromides are very strikingly homologous in their chemical and physical properties, and I cannot but think the efficacy of the two latter is due to their replacing, as it were, with greater energy the commoner chloride. The fact that large quantities of the chlorides are present in active changes of tissue—the healthy development of cartilage or the uproar of those changes which are rapid and extreme enough to be called inflammatory—is a very significant one. It leads us to consider what share the special crystallising and dialysing powers these substances may have in cell formation and nutrition. I do not see why we should not spend time in watching the changes in a piece of lymph on the iris when we have saturated the patient with an iodide, which salt we know does good, although we are not yet sure it replaces its congener the chloride. Then the different dialysing power of the chlorides of potassium and sodium, and the great differences in their physiological distribution ought to ensure to them also a competitive trial. The value of arsenic in nervous affections gives us

hints of this too. Part of the question of what I would call "Substitution Nutrition" may be investigated in health of plants as well as in disease of higher organisms.

I submit that these speculations, if they have any value, are most in place in a journal of ophthalmology; for ophthalmic surgeons not only see disease of the retina, choroid, &c., but they can *watch* changes in nervous, &c., tissues.

Professor Odling, in his most valuable and delightful "Lectures on Animal Chemistry," has considered the question from his point of view. The following extracts must not be considered as giving a full idea of Dr. Odling's opinions, but they may draw attention to his own more complete account of them. He writes: "It will be found, I think, that those mineral substances which act more especially as alteratives, actually are, and necessarily ought to be, characterised, not by their chemical energy, but by their chemical mobility; and I do not know that I can make my meaning better evident than by directing your attention to the chemical properties of iodine in comparison with those of its intimate congener, chlorine. As I shall presently show you, both elements possess in a striking degree the property of oxidizing various substances which resist the action of ordinary oxygen." P. 146–7.

"Now, while chlorine may be compared to peroxide of hydrogen, it is the sort of chemical mobility manifested by the oxides of nitrogen, which is characteristic of iodine, and, I believe, of most mineral alteratives. Iodide of hydrogen or potassium is, like nitric oxide, a facile reducing agent, and free iodine or hypiodite of potassium, like peroxide of nitrogen, a facile oxygenant—so that wherever the iodine travels it is capable of influencing the processes of oxidation there going on—absorbing oxygen where there is excess, delivering active oxygen where there is deficiency—just as our nitric compound absorbs oxygen from the air, and delivers it up to the sulphurous

acid. It is this chemical mobility of iodine, then, which chiefly distinguishes it from its more active congeners, chlorine and bromine. The general chemical relationship of these three elements to one another is most striking." P. 149–50.

APPENDIX OF CASES.

THE following cases are numbered, so as to follow in order the cases related in former papers I have published in this Journal:—

CASE XX.—*Epileptiform Seizures, with Temporary Defect of Sight, followed by Paralysis and Amaurosis—Syphilis.*

This patient (see also Case XVIII.) sometimes stopped his fits by a ligature. This seems to show that although the motor symptoms are chief, the sensory nerves of the skin or the muscles can influence the centre, possibly through its blood-vessels. As I have several times mentioned, I think the vaso-motor nerves are important links in determining the methodical expenditure of force which structure has acquired by nutrition.

George W., æt. 30, was admitted an out-patient at the Metropolitan Free Hospital in the autumn of 1862 for fits. He said that, whilst riding in an omnibus, a few days before, he had a quivering in the left side of the tongue and left side of the cheek. At the same time the eyes became "dim and sparkled." This continued for seven minutes, and then, he having left the omnibus, found that the left arm "was pulled right up." Next he slipped down, and became insensible, he thinks, for about half an hour. He could tell nothing about his condition in the full fit. In a little time he was able to walk from the police station, where he had been taken, to a cab.

Six weeks before he had had a little "working" of the left side of the mouth for about ten minutes. This was the only suspicious symptom of nervous disorder until the attack for which he came to me.

His general health was good, except that he had pains in the

limbs, and these pains were worse at night. For them he had been attending, as an out-patient, under Mr. Hutchinson's care, who had given him iodide of potassium. Two years before he had had chancres, followed by buboes. He had, however, had no other symptoms of secondary syphilis.

He did not attend again, but as I wished to know what had become of him, I sought him out, May 1863, and found that he was blind of the right eye completely, of the left partially, and he had paralysis of the left side of the face and of the left arm. The leg was scarcely affected. The facial paralysis was limited, as it usually is in the ordinary form of hemiplegia.

I learned that a week after I had seen him he had had another fit. His face was black, and he bit his tongue. Next day he was sleepy, and kept his bed. The left arm was useless for several weeks, and he lay in bed in a semi-insensible state. Since he had had no decided fits, but he had had little attacks of spasm of the face, of the left side. The optic discs were white, badly margined, and the vessels seemed to be strangled in it. There was now, I found, a node, tender and fluctuating, over the right parietal eminence. This soon went away with the iodide of potassium in ten grain doses, and, except for the blindness, he got into good health again. I admitted him into the Metropolitan Free Hospital, and during his stay he had one attack of spasm of the face, affecting, he showed me, only the part of the face which was paralysed. The paralysis gradually diminished, and he went out, except for the blindness, in good health.

This patient then went to an ophthalmic surgeon, Mr. Hulme, at the Central London Ophthalmic Hospital. Mr. Hulme had the great kindness to send him to me at the Hospital for Epilepsy and Paralysis in 1863. The patient was still blind of the right eye, but the sight of the left had much improved.

January 1865.—He came to me at the London Hospital for an ulcer two inches in diameter on his sacrum. It looked, from position and from its general shape, like a bed-sore. Under the iodide in doses of fifteen grains three times a day, it healed up in about two months.

On March 25, he said he had had a fit. He first had a *sparkling before his eyes*, then a twitching in the left hand, then fell, and became insensible; he bit his tongue. A few days later he had an attack, which affected sometimes the left hand, and

sometimes the left side of the face, and for two hours it prevented his keeping his head still on his pillow. He had a numbness of the left side for several days after. He several times had twitching of the left hand.

After this he occasionally complained of various odd feelings in the left hand, twitches, and of a sensation of pins and needles, and sometimes of flashes of light before the eyes, but these flashes of light came on at different times to the twitching of the hand. He has no defect of smell.

He had also occasionally pain in the neck and head enough to prevent sleep; and there was some tenderness of the scalp, but no node.

October, 1866.—He is still under my care for occasional slight and partial fits.

CASE XXI.—*Amaurosis, Anosmia, Cophosis—Subjective Sensations and Convulsive Attacks.*

Betsy Smith, æt. 18, sent to me by Mr. F. M. Mackenzie, was admitted into the London Hospital, March 25th, 1866, with loss of smell, blindness, deafness, and a certain difficulty in walking.

She had been ill two years, and began by "staggering and reeling about like a drunken person." She said she was not giddy then. About this time pain in the head began, and she has had it, on and off, ever since. It was at the vertex and front, and has been in the occipital region, when the blindness occurred. She commenced to be subject to vomiting, when the headache began, and this has continued. Now, when the pain comes on bad, she turns pale, feels sick, and gets up to retch.

I can get no very precise dates. The blindness came on about a year after the staggering, and was complete in about a year. The sight of the left eye went first. It is worthy of note that before her blindness she had temporary losses of sight. It would leave her suddenly in the street. When I first saw her, both optic discs had recovered from an acute stage of optic neuritis; they were anæmic, very loosely margined; the veins were irregular and dark, and some were partly buried on the disc itself. The arteries were visible, but they were very small. In the left eye the appearances were nearly the same. The

patient had lost smell, but for how long was not known. She had then subjective sensations of smells of various sorts; sometimes "like a drain," and sometimes "like spice." These would come on and off, and would sometimes last for a day. She could taste quinine and sugar, but could not recognize peppermint. She is quite deaf of the left ear, and partially of the right, and complains of great noises in her ears.

Now, as to her limbs, she could not use her hands well, and could, when I first saw her, neither extend her fingers completely, nor grasp firmly—indeed the grasp was very feeble. A few days later, when taken into the hospital, she grasped well. Then her hands were warm; before they had been very cold. Her walk, however, shewed the most striking symptoms. She could not walk at all by herself, and when supported, the right leg, she said, dragged. She shuffled it along. When we tried to leave her standing she clutched hold of us, and when quite freed she reeled, and would quickly have fallen unless she had been caught. If carefully balanced she could stand. Her gait was not "staggering," as she had called it, but a swaying, reeling, helpless progress, and was very unlike the staggering, eager walk of locomotor ataxy. Her general health was good.

She was taken into the hospital, and was, by the courtesy of Dr. Parker, placed under my care March 31st. She was in the hospital about one month.

On April 12th she complained of queerness before her eyes, and said "the place just now looks quite white." She said that just before it looked like straw; and, again, "like bubbles of water when the sun was shining." At the same time she spoke voluntarily of a sensation of smell in her nose. These sensations were altogether subjective, and she could neither see nor smell at all. The nurse said the girl wandered, but she seemed to me to be quite sensible, and so far as I could make anything out of the nurse's statement there was nothing to show that the girl's mind was defective. She was quiet and gentle. I mention these observations of the nurse who, unfortunately, was not an intelligent woman, as the girl's stepmother had several times asked me if her daughter was not very likely to go out of her mind. She gave as one reason for asking the question that the poor girl had threatened to commit suicide. Some time later she asked me the same question, but

gave very vague reasons for asking it. The girl was inquisitive; she asked the colour of my hair that she might distinguish me from my namesake, Dr. James Jackson. She was learning to read by a blind book very well.

April 21st. This morning, for twenty minutes, the right arm and leg kept moving. I did not see it, but I judged from the description that it approached tremor rather than cramp. Her speech was not affected, but she said she sometimes lost her speech. This loss must have been very temporary as her mother had never noticed it.

These are the only noteworthy circumstances which happened during the patient's stay in the hospital. Her general health seemed good. She was on full diet, and would, I was afterwards told, eat part of the diet of the other patients as well as her own. I did not then know of this, or I should have given her more food. Shortly after she left the hospital she had a fit. Her mother gave me the following particulars:—At tea one day the child got up to open a door, but suddenly cried out, "Oh, mother, take hold of me!" and then fell to the ground, and the right arm and leg moved, and the mother could not hold the hand still. The girl then cried out "Oh! what a stink! what a dreadful stink in the place!" and then, "Eh! eh! eh!" very loudly, and next went into a sleep and slept for hours. When she came to herself she knew nothing of what had happened, and answered questions.

[*May 7, 1865.—Betsy S., æt. 17, blonde, stout during last ten months; frontal headache and sickness in morning. Deaf and drowsy. First menstruated at 15, once only, and not again till two weeks ago, when after some treatment at St. Bartholomew's Hospital she had some bloody discharge during one day.

Unsteady in walking, and says "my legs give under me."

V. oc. d. qt. pt. oc. s. = No. 2 Jaeger's types.

Ophthalmoscopic signs:—

Oc. d. Optic nerve tumid, tissue hazy, vessels at exit hidden.

* This patient has been under the care of Mr. J. W. Hulke, at Moorfields, and under Dr. Stewart, in the Middlesex Hospital. To Mr. Hulke I am indebted for the above notes from his case-book. Unfortunately, the book in which Dr. Stewart's clinical clerk had made notes of this case was not to be found.

Contour of disc hidden. The opacity of nerve tissue extending into retina = $\frac{1}{3}$ or $\frac{1}{2}$ diameter of disc, beyond this retina clear, its veins swollen and tortuous.

Oc. s. Tissue of optic nerve hazy, not so much as to hinder the contour of the disc, but yet enough to make the central vessels (trunks and primary branches) very indistinct; nerve also tumid.

March 20.—V. oc. d. = qt. pt.; oc. s. = No. 20 Jaeger.

Deafness > Staggers in walking. Headache < Patches of greyish exudation in choroid.

1865. July 13.—Soon after last date she was admitted into Middlesex Hospital, under care of Dr. Stewart, where she caught typhus. Now she is very pale and thin. V. both eyes pt. = o.

Walks less unsteadily. No headaches. Optic nerves tumid, pale, woolly; vessels less hidden, and less swollen.]

November, 1866.—The patient is now, my friend Mr. Frederick Mackenzie tells me, in the Homœopathic Hospital.

Case XXII.—*Epileptiform Seizures affecting the Right Side—Amaurosis—Hemiplegia of the Right Side.*

Ellen S., æt. 43. This woman was placed under my care by the permission of my colleague, Dr. Davies, Sept. 22, 1866. She had then been in the London Hospital four days. The following history I obtained from her and from her daughter. The mother, although she would readily answer questions, soon became confused, and she gave very different dates at different times.

She had been ill since Christmas, 1865–6. The first thing observed wrong was a fit. She was taken, whilst washing clothes, with a pain in the right arm, "as if it were numbed." The arm next became stiff, and could not be moved, but the face and leg were not affected. This state of things continued for an hour (?), and the daughter went on rubbing the arm all along. Next, the mother made a noise in her throat, and soon after passed into the full fit. She was in this fit—doubtless a succession of fits—from 7 p.m. to 1 a.m. Next day she could use the arm, but she lay in bed six weeks. She kept her bed because she was "very ill," and I could get no exact account of her state. She had much pain in the left side of her head, but no vomiting and no paralysis.

She had no other attacks for two months, when she had the second. She had seven or eight at intervals; sometimes two a day, all of the same sort, before she lost the use of her right side. In some she bit her tongue, in others she did not.

The hemiplegia followed one particular fit. When the fit came on she was at her tea, and it, like the others, began by a stiffening of the right arm. After this attack she could not talk at all for fourteen days, but then she became able to talk.

From that fit to admission she remained hemiplegic, but the fits continued; and she had seven or eight before admission, each still affecting the right, the now paralysed, side. In about four or five of her fits—but in which of them I could not learn—she had been insensible.

After one of these, ten weeks ago, she lost her sight; for one week it was lost completely, but it then improved. After another fit, five weeks ago, she again lost sight, and never regained it.

The above notes date from the day when I first saw her. She was then lying in bed, hemiplegic of the right side, and blind. There was rigidity of the hand but none at the elbow or shoulder joints. The hand was shut, the last phalanx being extended, and the nails with their edges being pressed against the palm. The hand seemed the part most paralysed; she could raise the whole limb—not merely swing it from the shoulder—pretty well. I could not, with moderate force, open the hand, and pulling at the fingers gave her pain. I could make out no loss of sensation by ordinary tests, but I did not use the compasses, as although she could talk she was not quick. The leg was much paralysed, but she could just manage to get along without help. The leg was flexed at the knee, and could not be extended. The face was normal.

She could smell peppermint. She was quite blind. Both optic discs were white and slightly swollen; their edges were ill-defined; the arteries were thin and nearly lost in the disc; the veins were dark and irregular, but they were not very large. She complained of pain in about the mid-parietal region of the left side of her head. This pain did not extend into her face. She seemed in fair general health, and took her food well.

As to the nature of her disease I could not be sure; I felt certain that there was some coarse disease of the left hemisphere,

but of what sort I did not know. I, however, gave the patient the iodide of potassium in ten-grain doses, but not with much hope. The woman's daughter had an ulcer of the leg, which seemed to be undoubtedly syphilitic, but possibly it had been acquired from her husband, and was not the result of congenital taint.

She had a fit in the hospital on the 22nd, and the nurse told me that it lasted from 4 P.M. to 7 P.M. The nurse said the movements were strictly confined to the right arm, leg, and side of the face. The eyes "kept shutting and opening." It was said that the patient was not at all insensible. I asked the nurse if the patient talked. The nurse said no. The patient here eagerly put in a remark, that in her fits "she tried all she could to talk and could not." She added that she could always hear what was going on, and mentioned something that had been said when she was in the fit. However, it is not quite certain that she was conscious throughout. Although she could not talk voluntarily, she did get out the words, "I recollect," twice. She was, no doubt, then partly unconscious. On such secondary-automatic utterance I have spoken at length.—Med. Times and Gaz., June 23, 1866, Jan. 28, 1865; Lancet, Feb. 17, 1866. Lond. Hosp. Rep., vol. i. (See also p. 306.)

The patient was in the hospital about two months, and improved in her limbs a good deal. She could move the whole arm more, and was able to walk much better. The hand, however, remained, practically, useless.

It is quite as likely that the improvement in this case was due to time, as to the iodide. However, the patient improved after it. I have now under my care a patient, who leaves the London Hospital well after Christmas, although he was carried in totally paralysed of the right side two months ago. He took no drugs. No doubt, however, this man had a small clot.

CASE XXIII.—*Case of Loss of Speech, with Hemiplegia of the Right Side—Optic Neuritis—Death—No Autopsy.*

Richard A., 51, was admitted under the care of Mr. Adams for coma October 25th. He had fallen on a scaffold, and it was supposed that he might have received some injury to the head. As the coma cleared away, it was quite evident that the case

was a medical one, and it was believed that the man had fallen because he had had an attack of cerebral hæmorrhage. He was hemiplegic of the right side, and speechless. By Mr. Adams's permission I saw the patient November 24. The man was then, I believed, quite conscious, but he could not talk. He said nothing but "wo," "wo." (Not oh, oh.) He did nothing I told him. He simply stared at me when I spoke to him, and then looked away, still staring, with elevated eyebrows. I was told that he would put out his tongue, but he did not do it for me. The nurse said the patient sometimes smiled. He could eat well, and he could swallow well, as I ascertained by watching him eat a piece of dry bread. The nurse said he never asked for anything, and when he had eaten what he wanted he pushed the rest of his food away. He sometimes got angry, and the nurse fancied that he tried to swear, but he made use of no actual words in the process. He passed his motions under him, and took his fæces in his hands, to put out of bed as the nurse imagined. The man's hands were daubed when I saw him. I managed to see his optic discs. They were normal.

December 4th.—He has been removed to the Attic, and is under the care of Dr. Fraser. The patient still rarely or never says anything but "Wo, wo." Mr. Le Rossignol, to whom I am indebted for much courteous assistance in investigating the case, once heard him say "better."

I now beg particular attention to another matter. The nurse told me that one day, after his son had been long urging him to say if he knew him, the patient uttered this sentence, "How is Alice [his daughter] getting on?" The nurse could not understand the words, they were articulated so badly, but the man's son was certain that the sentence I have mentioned was really delivered. He told me too that his father had given him to understand where his tools were. He asked him the question, and the reply was "master's." This was all that he had ever said.

December 7th.—Nurse says he gets into passions and shakes his left fist at her, and looks vexed, and that he once tried to knock the candle out of her hand. She says he never makes signs for anything, except perhaps that when he wants anything he claps his lips. After much urging, and on seeing the nurse do it for his imitation, he put out his tongue. Generally he opens his mouth instead.

January 13th.—He is in the same general condition—still in bed, and still hemiplegic of the right side. I see him occasionally, and I can never get any reply from him. To use a common expression, "I can make nothing of him." Yet my friend Dr. Woodman, for whose opinion I have the greatest respect, says that for five successive mornings, the man has told the days of the week, *e.g.*, he held up one finger on Monday, two on Tuesday, and so on, but this morning he failed. The noise the patient can make has changed to Pooh! pooh! Indeed, he is generally called the "pooh! pooh! case."

When I saw the man first, that is in November, his optic discs were, as I have said, normal. I then saw them pretty easily, as he took little notice of what I was doing. I examined them again January the 14th, and found that he had well marked neuritis in both eyes. The difficulty in getting a good look at this patient's optic discs is almost inconceivable, and yet, although annoying, it was very interesting. It was, of course, necessary to dilate the pupils, and thus sometimes I could catch a glimpse of the discs. The common formula, "Look at my little finger," was of no use here. When told to do so the patient opened his mouth, or screwed up the eye, or moved his face to one side, or turned his eyes in several directions. I then asked him to look straight forward, but with no better result. I told him to take no notice of me, but this made no difference. Here was a curious state. He knew I wanted him to do a certain thing, but whether ke knew what thing, or whether, knowing it, he was unable to do it, I am not sure. I think it was probably a loss of voluntary control, as in cases in which patients do not put out their tongues when told, there is no ordinary paralysis, and they know what is wanted, as they put their fingers in their mouths, as if to get out the tongue. [On this part of the subject I have written, London Hospital Reports, vol. i, p. 400, and Medical Times and Gazette, June 23, 1866.]

Soon afterwards the patient left the hospital still paralysed, and still speechless. I called on him once, but his wife was out, and I could make nothing of him. I had no light for the ophthalmoscope. The man only said "Pooh! pooh!" Afterwards, my friend, Mr. E. Vialls, went to see him twice. The last time was on September 24th, and then Mr. Vialls learned that the patient had died three months before in a workhouse.

Of course I regret bitterly that I lost the opportunity of adding to the report of the case the facts an autopsy would have given. The case, however, is worth reporting incomplete as it is, if only to show the necessity of some organisation to prevent our studying disease in the fragmentary way we do. The co-operation of those who have charge of patients in workhouses is most desirable in helping ophthalmologists and physicians to follow out their cases. This is especially so in cases of Locomotor Ataxy,* a disease in which the ophthalmologist—I now mean for physiological reasons—is far more directly interested than the physician is.

I have (Ophthalmic Review, April, 1866, p. 52) spoken of the necessity of the study of Locomotor Ataxy by physicians and ophthalmic surgeons. In this group of symptoms there seems to be but one idea throughout. I am under the impression—a mere impression—that it begins not unfrequently with paralysis of the fourth nerve; and paralysis of the inferior oblique dislocates the whole of the movements of locomotion. I think, too, that amaurosis, with a gradually whitening disc, is sometimes the precursor of the leg symptoms.

The other day Mr. Frederick Mackenzie and Mr. McCarthy made for me an autopsy on one of my patients who had died with symptoms of Locomotor Ataxy under my care. This patient had had paralysis of both sixth, both third, and double atrophy of the optic nerves. Mr. Lockhart Clarke has this patient's pons, cord, &c. Surely a case of this sort ought to be studied by ophthalmologists.

I would at least suggest that ophthalmic surgeons should work in the physicians' wards. I have within the last two months had under my care in the wards of the London Hospital, thanks to the generosity of my senior colleagues,

* The questions of the relation of Sight and Touch and of the nature of our knowledge of Space, may, I submit be considered with advantage in these cases. If we are not born with intuitive ideas, we are born with complex anatomical possibilities, in such exact correspondence with our environment that we cannot determine how we acquire some of our notions, for we cannot remember when we were without them. The phenomena of disease seem to show that there are particular internal anatomical connexions betwixt sight and locomotion—that they are, to begin with, in a certain correspondence, so that the two experiences are readily integrated.

Dr. Davies, Dr. Andrew Clark, and Dr. Ramskill, cases which show the importance of doing this. A man, age only 20, with Bright's degeneration of the retina, who consulted me for fits; a man hemiplegic of the right side, and with great defect of speech, who has double optic neuritis; two cases of atrophy of the optic nerves in patients who have had fits of some kind; a boy who had double optic atrophy, and many other symptoms from disease of the crus cerebri and other parts; a girl with paralysis of parts of each third nerve and hemiplegia; a girl with hemiplegia, keratitis, and malformed teeth. Then, for negative facts, there were several cases of chorea. I and my namesake, Dr. James Jackson, yesterday examined five patients the subject of chorea, and found nothing wrong with their optic discs. We examined also four patients the subjects of unilateral epileptiform seizures, and found irregularity of one vein in the eye of one of these four patients.

I have also had under my care a woman the subject of unilateral epileptiform seizures, pains in the head, and vomiting. Her pain was from cranial nodes. There was nothing whatever the matter with her optic discs, except perhaps that they were a little pale, but I mention her case, as it is, in my opinion, a very likely one for after-neuritis to occur in, for it is important not only to recognize the slightest changes in the optic discs, but to recognize under what circumstance optic neuritis is likely to occur.*

Case XXIV.—*Subjective Sensation of Smell, with Epileptiform Attacks.*

According to the view I take that subjective sensations of all kinds should be compared and contrasted, the following letter from a patient now under my care is of interest in connection with Cases XX and XXI. It is only part of his case, and is inserted here merely as a hint of the necessity of broadening our studies:—

"Sir,—I have much difficulty in describing my first fit, which took place about two years ago, on account of my memory being

* She has since been re-admitted with optic neuritis. As a rule optic neuritis is to be feared when unilateral convulsions are attended by vomiting and headache.

so much impaired by the disease. I was standing by the press preparing a sheet of type for printing; suddenly a sharp smell crossed my nostril: I fell as though I was shot. I was told afterwards, by one of the men that picked me up, the paroxysm lasted about three minutes, during which time consciousness was entirely suspended. When I came to myself I could see the parties that held me. My first question was, what day of the week is it? where am I? Although I could see the parties holding me, I was not conscious where I was. They set me down, gave me some water, and in about twenty minutes I was going on with my work again.

"I felt no more for two months, when one day I felt very poorly. After dinner I laid me down on some chairs and fell asleep. After a short time my family was alarmed at my making a strange noise. They came to me and found me in a strong fit; my hands closed very tight, but no particular distortion of the face. In the course of that afternoon I had about eight fits. The effect produced by them was lowness of spirits, loss of appetite, partial loss of memory, and total loss of smell.

"Four weeks after this I had another, which came on soon after going to bed. I had gone to sleep; I felt a sensation in my head like a squeezing of the brain; it was momentary. I was not conscious again till next morning; whether I had been in a sleep all night or in a state of coma I could not say; my wife said she could not awake me. The effect produced was much the same as before. I continued to have them periodically, sometimes two or three days over the month, and sometimes the reverse, up to two months ago, from which period I had an interval of seven weeks and three days, which brought me up to last Sunday week. Between five and six in the morning of that day I was between asleep and awake, when I felt the paroxysm coming on me. I had no power to move; I made a strange noise; my wife tried to get me up to no purpose. I lay in this way about an hour when I felt it coming on me. I had the power this time to call out "It's coming on me again." This lasted about one hour. When I came out of it I could not remember the day of the week; in fact, my mind seemed to be completely deranged, which feeling I have had on me more or less ever since. It all seems to proceed from the upper part of my nostril; a strange, indescribable smell seems to communicate with my brain. This attack has

been more severe than before. I felt it from my head to the bottom of my feet."

Postscript No. 1, *referred to p.* 284. — The following is a quotation from a report in the "Mirror of the Lancet" (Nov. 26, 1864, p. 604), of some remarks I made on "Defects of Expression," &c. First mentioning that the differences in defects in patients who have disordered speech is probably due to differences in the "quantities of brain damaged about the highest part of the motor tract, the corpus striatum—the point of emission of the orders of the 'will' to the muscles," and that damage to the brain in the neighbourhood of the *right* corpus striatum does not produce loss of speech in any sense of the word; I was correctly reported further as follows:—"Now amaurosis is due to disease of *a highly specialized nerve of sensation*, whilst there are good reasons for believing that defect of articulate language, which M. Broca calls aphemia, is a kind of ataxy of articulation, and, therefore, *a disorder of motion.* Again, the contrast may be made more general. Sight is a department of general perception, whilst articulate language is a department of general expression. If, then, it should be proved by wider evidence that the faculty of expression resides in one hemisphere, there is no absurdity in raising the question as to whether perception—its corresponding opposite—may not be seated in the other."

Postscript No. 2, *see p.* 290.—Now it is in some classes of cases of disease of the nervous system "hard to say where obviously motor symptoms end, and where the purely mental ones begin. Thus there is (in cases of hemiplegia on the right side) every gradation betwixt, on the one hand, a total loss of the power to express ideas, or a loss of knowledge of the relations of words to things, and, on the other, apparently scarcely more than a peculiar motor defect in talking—an ataxy of articulation. And sometimes in the same case we find that the patient makes mistakes in words, and also articulates badly. In the case of the subject of these remarks there are (1), involuntary ejaculations; (2), involuntary and yet complete actions; and (3), local spasm or twitching of muscles. We may, perhaps, find all shades, degrees, and analogies betwixt obvious and coarse motor reflex actions, and disorder of what Dr. Laycock has described as the reflex function of the brain. We may thus analyse with some success, from the study of phenomena which are superficial and simple, more hidden and intricate mental conditions.—"Remarks on a Case of Involuntary Ejaculations—Chorea," Med. Times and Gazette, January 28, 1865, p. 89.

ON A GROUP OF CASES OF OPTIC NEURITIS IN CHILDREN.

By Jonathan Hutchinson, F.R.C.S.

The cases in which blindness results from optic neuritis may be divided into several distinct groups. In some this disease is due to arachnitis, in some to cerebral tumours, and in others to blood poisoning. Several other less frequent causes might be mentioned, and under the term blood-poisoning we might probably instance several different kinds, *e.g.*, syphilis, the specific poisons of some other exanthems, and perhaps some minerals, such as lead.

My attention has for some time been drawn to a group of cases in which optic neuritis occurs in children, and in which the symptoms differ in some respects from what we usually find in others. Nearly always there is a history of a severe illness, which was supposed to be fever, and was marked by delirium and other head symptoms. As the child recovered from this it was found that blindness, either partial or complete, had occurred. Frequently such children regain good health, in fact recover perfectly, excepting as regards the sight. Hemiplegia, or any other form of paralysis, very rarely occurs, nor does the intellect suffer. There appears to be but little tendency to relapse. In these features we have much difference from what is usual in neuritis in adults.

These cases appear to me to be so peculiar, that I have thought it worth while (with the able assistance of Mr. Waren Tay) to collect those of which I have notes into the following table, in the hope of placing their features of resemblance and of difference in a clearer light.

In many instances, owing to the age of the patient, the ophthalmoscopic examination was made under difficulties, and was not so complete as could be wished. I

have noticed that in a few cases, not only is there evidence of neuritis, but also of peculiar changes at the yellow spot. Although there are rarely any indications of general retinitis, yet at the yellow spot itself there is sometimes seen a group of highly refractive globules, resembling at first glance a cluster of spider's eggs. These groups are almost symmetrical, and are very definite. I have not often seen anything resembling them in the eyes of adults. A case is published in an early volume of our Reports, with a coloured drawing, in which they are represented. Dr. Bader there describes them as looking like the vesicles of herpes, and the comparison is really apt. In that instance the subject was an adult man, who had become blind after an illness attended by vomiting, pain in the head, &c. The conditions were symmetrical, and there was no clue to any special cause.*

In a few of my cases the proof that neuritis had occurred is not positive, the patient having passed into the stage of advanced atrophy before coming under observation. Judging, however, from their close resemblance, both in history and in general features, to the other cases, I feel no doubt that a stage of neuritis had preceded the atrophy.

My cases appear to have special value as regards the prognosis of this disease, since in several of them I have been able to extend the history over some years.

I would venture to ask attention to the fact that cases of primary white atrophy, without head symptoms and without assignable cause, such cases as we so frequently meet with in adult men (tobacco amaurosis) are unknown in children. In almost all cases in children there is good reason to believe in neuritis as the first disease, and in the few which I have seen of primary atrophy, there was a clear history of syphilis.

* See Ophthalmic Hospital Reports, Vol. II, April, 1859, Plate viii, figs. *a*1 and *a*2.

TABULAR STATEMENT OF CASES OF OPTIC NEURITIS IN CHILDREN.

No.	Name, Date, Reference, &c.	Age.	State of Health on Admission, &c.	History of Former Illness.	State of Vision of Pupils, &c.	Ophthalmoscopic Examination.	Remarks.
1	Julia Garner. p. 38 B. April 17, 1862.	11	Head thrown back. Cervical muscles rigid and somewhat tender on pressure. Her mother thought this had been always so. Tongue clean. The pains in the head (back and front) and neck had subsided, but had returned a few days before.	Very restless nights. Much headache. No history of diphtheria, or of any exanthem. No failure of power in arms or legs. She could see well till three months before, when she began to grope her way about. At night she had flashes of light. No muscæ. She lived in an ague district. For six weeks previously her sight had been much as on admission.	Pupils of moderate size; exceedingly sluggish; almost motionless. Left slightly larger than the other. Aspect decidedly amaurotic. Face pale; placid. She could just distinguish the large bars on the windows. The globes twitched, but there was no paralysis of any muscle. The eyelids, possibly, drooped a little.	Condition in both eyes very similar. Media quite clear. Optic discs not well defined, and of a grey pink tint (not white). In the left it was impossible to distinguish the margins of the optic disc at all. The arteries of retina were very small, but the veins very large. Extending from the optic disc in each eye towards the yellow spot was a large group of small brilliantly white patches. Some of them very small; others rather larger; all round or oval. They resembled the perforations of a worm eaten leaf or a group of butterfly's eggs.	She died in the Blind School, London, April, 1864, where she had been for a year. A month or two before her death I saw her. She was then in good health; quite happy; walked well. Venæ centrales large and conspicuous. Arteries small; discs very white. Pupils large and motionless. Divergent squint. Dr. Sharp informs me that she died with cerebral symptoms.

TABULAR STATEMENT OF CASES OF OPTIC NEURITIS IN CHILDREN—*Continued.*

No.	Name, Date, Reference, &c.	Age.	State of Health on Admission, &c.	History of Former Illness.	State of Vision of Pupils, &c.	Ophthalmoscopic Examination.	Remarks.
2	Susannah Burrows. p. 132 B. Sept. 15, 1863.	10	A stout, flabby, pale child. Could walk well. Pain and sickness had ceased six weeks before. She was cheerful. Hearing good. Slept well. She had no cough. Both parents living and healthy.	In April she had scarlet fever, and was very ill. Afterwards she had œdema of the legs (slight). The left eye was the first affected. In about three weeks she was quite blind. She had pain in her head (chiefly frontal), and vomited. She never had "fits."	Total inability to perceive light. Pupils large; of equal size, and almost motionless. The left globe looked outwards. Aspect amaurotic.	Conditions similar in both. Media perfectly clear. Optic nerves white (very); veins large; arteries very small; choroid orange-red, not markedly so. No other changes.	In this case the pain would often last several days and nights, and prevent her sleeping. The vomiting occurred once in two or three days; not at any particular time. No failure of muscular power.
3	Louisa Butler. p. 173 B. March 17, 1864.	12	A very pale, delicate - looking child of amaurotic aspect. Could walk well, and had power over sphincters. Never sick; no pain; cheerful; appetite good; could sleep well; was gaining flesh	Three months ago she had a severe illness, with most severe headache and vomiting. She was somewhat delirious, and would scream much. Dec. 10.—Admitted into St. Bartholomew's. Dec. 23.—Quite sensible, but motions passed involuntarily. Could not move head well.	When she left Bath she could see very fairly. She believed she could read small print. About a fortnight afterwards her sight failed rapidly, and in a fortnight she was quite blind.	Great difficulty in getting her to fix her eyes. Conditions almost perfectly similar in the two eyes. Optic disc swollen, elevated, greyish, and woolly looking. Veins tortuous; everywhere visible, but as if in a greyish semi-transparent substance. Arteries much diminished,	Detailed notes of her illness whilst in St. Bartholomew's have been kindly furnished to me by Mr. Glynn, Clinical Clerk to Dr. Jeaffreson. 1864. March 22.—Urine pale. Deposited lithic

			rapidly ; she could move her head freely.	At time of discharge, Jan. 14, her motions and urine passed involuntarily, and she could not walk. She soon regained power.	Pupils widely dilated and fixed. Weakness of right int. rectus.	but still to be seen. No extravasations.	acid. 1866. Dec.–Inmate Blind School (Brighton). Quite blind, but in excellent health.
4	Robert Loamy. p. 178 B. January 15, 1866.	1 yr.	The child was pale. The anterior fontanelle was open. Feb. 23.—General health improved. March 29. — In good health, and very cheerful. The anterior fontanelle still large, but the head not hydrocephalic. June 4.—In good health.	Supposed to have been blind several months. The mother first suspected it three weeks ago. She considered that he could see well till he had "fits" in connection with teething. He was noticed to squint after the fits. He had four "fits," during which he turned quite stiff, and clenched his fists. He could always move his arms and legs.	Feb. 23.—Appeared to be blind. March 29. — The mother thought that he did not see at all. His pupils were of moderate size, before the use of atropine, and dilated well with it. Aspect not amaurotic ; indeed, he appeared at times to be fixing objects ; but he did not appear to see in the least.	Discs grey-white, of a dirty colour ; the margins indistinct and blurred. Arteries small, veins rather large. The retina everywhere, except close to the disc, transparent and clear. Feb. 23.—Much the same. March 29.—Discs clearer, and not very pale. The retinal arteries small, but the veins of fair size. The margins of the discs tolerably distinct, and the atrophy not nearly sufficient to account for complete blindness.	A grain of iodide of potassium was ordered three times a-day. No syphilitic history obtainable. June 4.—He can now certainly see. He will seize large objects held before him, but slowly. Expression of eyes decidedly vacant. Dec. 17.—Can see much better.
5	Mary Chitty. p. 125 C. Dec. 6, 1866.	6½	Good.	The left had been failing since an attack of measles at age of twelve months. She had no fits. The right		Both discs pale and indistinct. Right disc a little elevated, and trunks of vessels on its surface somewhat con-	

TABULAR STATEMENT OF CASES OF OPTIC NEURITIS IN CHILDREN—*Continued.*

No.	Name, Date, Reference, &c.	Age.	State of Health on Admission, &c.	History of Former Illness.	State of Vision of Pupils, &c.	Ophthalmoscopic Examination.	Remarks.
				had failed for a fortnight only.		cealed. Left disc flat, and less lymph about it. Vessels (artery and vein) diminished very considerably. The left ones the smaller. Choroid pale, with minute dots of white in front of it, ill-defined, and occurring chiefly in a group near the yellow spot, but also at other parts.	
6	Charles Hoskins. May 28, 1866. p. 14 C.	11½	A pale thin lad, a ravenous eater, but always thin. His father attributed this to worms. He had very imperfect power in his legs; he could just walk without assistance, but he seemed as	Two months before he was in Guy's Hospital for a severe illness. His legs were paralysed, and he was insensible for some time. He had very little pain in his head. The first symptom noticed was that he squinted. This passed off as he began to lose the use of his legs. He	He could not appreciate even the flash of light from the ophthalmoscope.	Optic discs anæmic. Vena centralis somewhat larger than natural, and the arteria decidedly smaller, but minute twigs can still be traced on the disc. The apparent inner side whiter than the outer. In the right eye the edge of the disc very indistinct, and the disc	Admitted into the London in June. While in he had occasional sickness, and when discharged in July his sight was still the same. After his discharge he became liable to fits, in one of

			if he would fall every minute.	had no convulsions.		itself looked elevated and swollen. In the left eye the margins of the disc were much more abrupt.	which he died on Nov. 27. No autopsy.
7	Charles S. Wood. p. 16 C. Jun 4, 1866.	11	A sturdy, healthy looking boy, quite cheerful; could walk well. His father considered that he had been a "weakly child." He complained of pain in the back of his neck.	When 14 months old he had "inflammation in the lungs, and was insensible." He got quite well. His sight was first noticed to fail three months after an attack of small pox. In September his father noticed that he did not write straight, and then he found that the boy could not see well. He had not complained of pain in his head.	He could only read No. 14 (Jaeger) at three inches; he could not make out anything at a distance; not improved by glasses. Sept. 24.—The father considered that his sight was improving; he could write a little on a slate.	Discs of a dirty white colour, the vessels on them of fair size, but somewhat obscured, and the edges of the disc a little irregular. Oct. 22.—Discs very clear; atrophy of nerve, very decided pallor, and some cupping; vessels of fair size.	On October 22 his sight was as at first note. Dec., 1866.—Much the same.
8	Wm. Fisher. p. 19 C. May 28, 1866.	13	He could walk quite well; when told there was nothing in the way he walked boldly.	History of fits and "dreadful pain in his head" in February. Was admitted into St. Thomas's Hospital (Dr. Bristowe) in March. At this time his mother said the right eye looked larger than the other. In the hospital	Both pupils motionless, the right being twice the size of the left. No perception of light from the ophthalmoscopic mirror.	In both the optic discs were dirty and fluffy, the margins being concealed; the arteria centralis much diminished in size, and the vessels on the disc being concealed at parts by the deposit of lymph. In both the	

TABULAR STATEMENT OF CASES OF OPTIC NEURITIS IN CHILDREN—*Continued.*

No.	Name, Date, Reference, &c.	Age.	State of Health on Admission, &c.	History of Former Illness.	State of Vision of Pupils, &c.	Ophthalmoscopic Examination.	Remarks.
				he got much worse, and was expected to die. When he was discharged he could not walk; he did not seem to have any use in his legs. She considered that he could see until the end of April.		appearances of neuritis were passing off, but the right was further advanced in atrophy than the left.	
9	William Henry Fowler p. 35 C. June 25, 1866.	7	He had been taking cod-liver oil, and was in better health than he had been for some time. There were scars of old strumous disease about his right carpus. He could walk quite well, and was very cheerful. Aug. 20.—He was in good health; every sense be-	Rather more than twelve months previously he had what the father called "sore-throat fever." He was insensible for a fortnight or three weeks; he lay on the bed not knowing anybody. He went to school afterwards, and his sight was not noticed to be defective at all. Sixteen weeks before admission his right failed suddenly. His father	For sixteen weeks he had seen daylight occasionally, but more often he could not; his pupils were equal, and acted fairly, but never contracted to small size.	Conditions symmetrical in the two eyes. Media clear. The optic discs had a definite outline, easily seen, and almost round. They were pale, and uniformly so. The vessels were diminished in size considerably. On the disc itself, although the trunks of the vessels were easily seen, they did not appear to have that distinctness of outline which is common in mere atrophy. I think	He was ordered a grain of iodide of potassium three times a day.

			lieved to be perfect excepting sight.	considered that he could see quite well one night, and the next morning when he got up he could not see his way about. "He had to feel for things." The week before he lost his sight he complained of pain in his head, but ran about as usual.		there must have been a stage of neuritis. At the yellow spot in each eye there were a series of small white patches, streaks and dots, some of them perfectly round. It is difficult to say whether these were from deposit or atrophy.	As he was quite blind, the examination was made under some difficulties. This condition of the yellow spot in each eye exactly resembles what I have several times before seen in neuritis in children, and very rarely in adults. During the illness mentioned above he had convulsions, and his hands were bent on the fore-arms.
10	Anne Rolf. p. 10 C. May 7, 1866.	40		"Water on the head," "fits," often fell down stairs in childhood. She stated that she was blind of both eyes for a month, but that then the right improved. The left continued blind.	Could not see to do needlework, but could see large print. She said — $3\frac{1}{2}$ made things look brighter, but there was no definite improvement.	White atrophy of optic discs. Artery and vein (central) in each eye easily seen. No things on the disc could be distinguished. Left more advanced than right. Discs looked small and irregular, the left especially so. In the left eye there	This case is introduced as showing (probably) the state of things long after neuritis (optic) in childhood.

TABULAR STEATEMENT OF CASS OF OPTIC NEURITIS IN CHILDREN—*Continued.*

No.	Name, Date, Reference, &c.	Age.	State of Health on Admission, &c.	History of Former Illness.	State of Vision of Pupils, &c.	Ophthalmoscopic Examination.	Remarks.
						were patches of absorbed choroid, with deposit of pigment.	
11	Henry Bayliss. p. 40 C. June 28, 1866.	7	Intelligent, active, fond of swinging, &c. Always "running about and playing." Taste said to be good. Smell very defective. Probably he cannot smell at all.	In 1863 he caught cold, and was taken ill with pains in neck shoulders, elbows. His mother could not keep him in bed; "he was almost mad." He was confined to bed for nearly three months, and every now and then had "fits." He always knew his mother. He never lost the use of his limbs, but when he began to get about he would stagger and fall down sometimes. It was then noticed that he could not "find things."	Pupils large and fixed. Globes divergent and twitching. He said he saw light for the first time for two years a few days before admission.		There was no clue to any syphilitic taint. Dec., 1866.—Much the same.

				Six months after the commencement of the illness he was quite blind.			
12	Charlotte Thompson. p. 73 C. Oct. 1, 1866.	7	Fair complexion, red hair, large head, thick alæ nasi. Could walk well, was cheerful, but nervous. Forehead prominent (like her mother's). Quite intelligent, mother considered that her head was injured by use of forceps.	The illness commenced on New Year's Day, 1866. Pains in limbs, sickness, convulsions, loss of speech for an hour or two at a time while recovering from the convulsions, delirium at times, complete paralysis of legs, and for long while inability to bend her back, stiffness and pain in neck. Always passed motions &c., consciously. At end of five weeks she began to get about again, and it was then noticed that the eyes were crossed. She was supposed to see well. Her hair all came off. In a week or ten days the left eye was found to be failing. In a month (March) the right eye failed also.	Large sluggish pupils. Could distinguish daylight, fire-light, candle-light, &c.	White discs (dirty white), abruptly margined. No disease at yellow spots. The larger veins and arteries somewhat diminished in size.	A grain of iodide of potassium was ordered three times a day. She is now in good health, but quite blind, and her mother is endeavouring to get her admitted into a blind school. (Dec. 4, 1866).

TWO CASES OF CYSTICERCUS IN THE EYE.

By T. Pridgin Teale, Junr., M.A., Oxon,

Surgeon to the General Infirmary at Leeds.

Although several instances of cysticercus in the eye have been already brought before the profession, the two following cases appear to me to be of sufficient importance and interest to justify me in recording them:—

Case I.—*Cysticercus in the Vitreous Humour.*

Martha Colbeck, of Batley, æt. 45, consulted me at the General Infirmary in the latter part of 1864. She complained of weakness of sight of recent occurrence affecting both eyes. She did not direct my attention to one eye more than the other. As she had a convergent squint, involving especially the right eye, I examined the eye carefully, and found the sight deficient. She then told me that she had squinted since infancy, that the vision of the right eye had been defective since childhood, and that this deficiency had not materially increased. The eye had never been inflamed nor painful.

On examination, I found that with the right eye she could, with difficulty, read 8 Jäeger, and that the field of vision was imperfect, but could not be mapped out in consequence of her inability to fix the eye steadily. Iris active, and equal in colour to the left. Aqueous clear. Tension normal.

Examination by Ophthalmoscope.—About the centre of the posterior surface of the lens there is a brilliant white spot, which is prolonged backwards into the vitreous by a dull, grey neck, which then rapidly swells out into a balloon-shaped body. The body is semi-opaque, bluish, slightly corrugated, and of such a size, that only about one-third can be seen in the field at one time. It recedes from the centre of the lens towards the nasal side. The vitreous is clear, and the natural reflection of the choroid is visible at the temporal side of the opaque body. By "oblique

illumination" the brilliant white head of the cysticercus can be seen at the back of the lens, the body being undistinguishable.

As the patient was not able to keep the eye steady it was impossible to ascertain whether any variations of shape of the animal took place, or whether there were any signs of vitality.

This patient was first seen in 1864; again in October, 1865; and, lastly, June 16, 1866.

During this period of two years no change has taken place in the appearance of the eye, or of the animal. The sight has slightly deteriorated, but the eye does not appear to suffer in any visible degree from the presence of the entozoon.

Two questions of considerable interest may now be raised. First, how long has the cysticercus been a tenant of the vitreous humour?

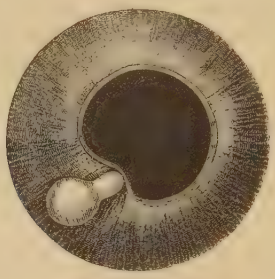
Since infancy there has been strabismus, and since childhood a defect of vision, which has continued till the present time with but little variation. There is no history of inflammatory disturbance which would point to the period of the invasion of the animal. Are we, then, justified in suspecting that the original defect of sight and the strabismus may have been caused by the presence of a cysticercus, and that the cysticercus has been in the eye almost from birth?

The second question is, whether removal of the animal is possible, and, if possible, whether it is desirable to make the attempt.

It seemed to me that it would be possible to introduce the suction-curette through a small opening in the sclerotica into the vitreous humour, and, whilst observing the movements of the instrument by the ophthalmoscope, to plunge the point of the curette into the body of the cysticercus and remove the fluid contents by suction. The body of the animal might then perhaps have been partially withdrawn by means of the curette, or left as a shrivelled membrane. Before deciding on any operation I wrote a description of the case to Mr. Bowman, asking his opinion; he replied, "Gräfe says your cysticercus is probably in the vitreous canal, and comparatively harmless from its fixity. Therefore he would not touch it, as it is of great risk of loss of eye." In accordance with this opinion I have refrained from any operation, and content myself with a periodical examination of the case.

Case II.—*Cysticercus on the Iris.*

Mary Isabel Bateman, æt. 10, living at Armley, was brought to me on June 2nd in consequence of tenderness of the right eye. On examining the eye there was seen (vide drawing), on



Pen-and-Ink Sketch (enlarged), showing the position and shape of the Parasite.

the surface of the lower part of the iris, an opaque body, constricted in the middle, and rather larger than an hemp seed, which was evidently causing some distress to the eye. The conjunctiva was slightly injected, the cornea was bright, but dotted on its posterior surface with minute spots, as in corneo-iritis; the iris was active except at the situations of the white body, near which it was adherent to the capsule of the lens. Tension normal. Reading No. 16 Jäeger. Her mother gave the following history of the white body:—

For two or three years the eye had been occasionally inflamed. Six weeks ago she first noticed a speck on the iris of the size of a pin's head. Two weeks later, the speck having grown larger, she consulted Dr. Hepworth, of Armley, who detected a bladder-like body, which he supposed to be a cysticercus. At the end of five weeks the spec was doubled in size, and had become constricted in the middle, after which the eye became dim and painful. On further inquiry I found that the child had always been delicate, and had long suffered from thread-worms, but not from tape-worm, or any other kind of worm. In August of last year she was attended by Mr. Scattergood in a severe attack of scarlet fever, with albuminuria and head affection, during which she had convulsions for two or three days followed by strabismus,

which afterwards disappeared. Since that time the right eye had been tender, and the right arm weak.

June 9.—An incision having been made at the margin of the cornea with a cataract knife, the piece of iris on which the animal was fixed was withdrawn, and cut off without destroying the cysticercus. When removed from the eye the slow movements of the body and changes of shape were easily detected. Examined by the microscope, the head and neck surmounted by the circle of hooklets, and four suckers were seen to project from the side of the body. (The rough sketch below.)

June 12.—The eye has been more comfortable since the operation, is free from pain, and the dots of lymph have disappeared from the posterior surface of the cornea.

14th.—The eye is free from redness and irritation except at the wound. Reads 16.

23rd.—Reads 16.

November 3rd.—Reads No. 1.

In selecting iridectomy in the treatment of this case in preference to any attempt to remove the cysticercus alone, I considered that two advantages would be secured. In the first place, iridectomy would accelerate the restoration to health of structures suffering from the presence of the cysticercus; and in the second place we should thereby avoid the almost certain prolapse of the iris; a complication by no means trivial in an eye already deviating from a healthy state.

NOTES OF TWO CASES OF COLOBOMA, AND ALSO OF THE DISSECTION OF AN EYEBALL AFFECTED WITH POSTERIOR STAPHYLOMA.

By Spencer Watson, F.R.C.S., Eng.,

Two Cases of Coloboma of the Iris, associated with corresponding deficiency in the Ocular Tunics.

Case I.—Mrs. L., æt. 30, married, applied at King's College Hospital on July 13, 1863. She had a nearly symmetrical deficiency in both irides at the lower and inner side. Her field of vision was curtailed above, but she was quite able to work with her needle. Under oblique illumination the pupils were observed to have a thin film over them, and the pupillary margin was noticed to be irregular and fringed with pigment at several points. The ophthalmoscope shewed a bluish white surface extending from near the cleft in the iris to within a short distance of the optic disc, which latter was normal in appearance, and separated from the coloboma by a narrow bridge of healthy choroidal tissue. Sclerotic vessels distinctly ramified on the white patch, and irregular islets of choroidal pigment were scattered along the edges of this latter. Both the eyes presented very similar appearances. She herself was an otherwise well formed woman, and she had several children, who were all free from any ocular defect.

Case II.—In October, 1866, a girl, æt. 18 years, came to the Central London Ophthalmic Hospital with her brother, who had a very extreme convergent strabismus. She was healthy and otherwise well formed, but had a deficiency in the lower part of each iris; that in the right eye being much smaller than that in the left. She stated that another brother had been operated on for squint, and that all her family had some ocular defect.* The right eye was very amblyopic, and only slightly improved by concave glasses. The left eye was decidedly improved by $-\frac{1}{20}$ for distance, and with this eye she was in the habit of working and reading, though with some difficulty. In both the field of vision was limited above. With the ophthalmoscope a white membrane was seen quite bare of choroid or retina, extending from the cleft in the iris quite up to the optic nerve in each eye.

* The brother has since been brought to the hospital, and he has coloboma of one eye.

The white tract was sharply margined by the choroid, and no bridge of this latter coat or of the retina intervened between it and the optic disc.

Dissection of an Eyeball affected with Posterior Staphyloma, in which some peculiar appearances were observed.

The eyeball was removed by Mr. Bowman on October 30, 1866, from a man 25 years old. It had been blind from early infancy, and exhibited the following appearances. Nearly the whole cornea was leucomatous, and there was total anterior synechia. The globe was much elongated in the antero-posterior axis, and with backward extension of the posterior pole, the optic nerve had been pushed very much inwards. A section of the fresh eyeball was first made through the equatorial region, and the interior of the posterior segment was inspected through the vitreous. At the nasal side of the optic disc, which was small and white, and slightly excavated, a white, woolly-looking oval body, about 2‴ in its long, and 1½‴ in its short diameter, was seen partly projecting into the vitreous humour. In a section made through this woolly body, after the globe had been hardened by immersion in spirits of wine, the nerve sheath was seen to be thickened at the nasal side, and the white body appeared as a membrane lying upon and co-extensive with the thickened sheath. The sclerotic at the posterior pole was thinned. Examined with a $\frac{1}{5}$-object glass, the white membrane exhibited successive layers of fine and large granular cells, having some resemblance to those of the normal retina. Where the opaque membrane was continuous with the retina, these layers ceased abruptly. No nerve fibres were seen with this power.

The close resemblance which this white woolly membrane, seen through the vitreous humour in the fresh state, bore to the appearances presented in cases of neuritis of the optic nerve, suggested the idea that some of these may possibly be pathologically allied to this case, which I am disposed to regard as an instance of thickening of a wasted retina, proceeding from chronic inflammation, transmitted to it from the subjacent sclerotic.

NOTES OF MISCELLANEOUS CASES.

By Jonathan Hutchinson, F.R.C.S.

(Continued from page 222.)

No. VII.—*Choroido-retinitis in Two Sisters, ending in Blindness in one of them at the age of* 17.—*Infantile Paralysis in the other.*

The two following cases are interesting in reference to the obscurity of the cause to which the choroido-retinitis was due. Although no history could be obtained, I yet suspect in the absence of other explanation, that there must have been a taint of syphilis. A few weeks ago, at one of our evening demonstrations, Mr. Streatfeild brought forward a girl with well-marked choroido-retinitis, whose central incisors were most typically notched.

Ellen White, æt. 17, an ill-developed, sleepy-looking girl, attended in the beginning of October, 1866. The history we obtained was that at school, when quite a child, it was noticed that she could see better at a long than at a short distance, but at 12 years of age she began to squint (convergent). Her mother took her to Guy's Hospital. The squint was operated on, and she was told she must wear spectacles. After the operation, for a time she was annoyed with double vision; this, however, passed off, and it was considered that she was much benefited; but before very long her sight began to fail, with occasional pain around the orbit, and inability to see at night, or in a dull light. She had no muscæ nor flashes of light. Her sight slowly got worse, until she became, as she is at present, quite blind. She had menstruated three times only.

Her mother had borne twelve children, ten of whom died in infancy. An elder sister (living) and our patient made up the number. The mother said that she and her husband were not related before marriage. No history of symptoms suspicious of syphilis (excepting the infantile mortality) was obtainable. The father had died after a long illness, attended by paralysis.

Ophthalmoscopic Examination.—The eyelids were half closed;

there was slight divergence of the left eye, and the pupils were of medium size, and quite motionless. The two eyes presented the same features. The discs were of a dirty grey white, and the vessels so small that they could only be traced with difficulty beyond the margin. There were but few denuded patches of sclerotic. The pigment was accumulated in "blots" rather than stellate patches. There were a great number of these "blots," and more of them near the centre than at the periphery. Many of the pigment patches were in the retina itself.

Her elder sister, Catherine (æt. 19), also came. She was the subject of infantile paralysis. Her sight had been failing for twelve years, and her eyes were in much the same condition as her sisters', but less advanced. She can still see large letters.

No. VIII.—*Slowly Progressive Blindness from the age of 9 years.—Retinitis Pigmentosa with the accumulations most abundant near to the disc.—Extreme shrinking of Retinal Vessels.*

The following case presents some features of similarity to the preceding one. In both the optic disc was atrophic, and the central vessels shrunk to extreme minuteness. In both the accumulation of pigment in the retina was very great, and chiefly near to the disc and yellow spot, not as usually observed in retinitis pigmentosa, most abundant at the periphery. In both the progress of the disease had been very gradual, and without assignable cause, and had gone on to complete blindness. In neither were any other indications of nervous disease to be discovered.

H. B., a moderately stout, intelligent man, æt. 52. He believes that he was quite healthy in early life, excepting that at the age of 4 years he had convulsions during measles. In childhood he learnt to read. At about the age of 9 he began to lose his sight. At the age of 11 he went to sea, but as he "could not see the masthead," they refused to take him as an apprentice. He returned home in nine months, suffering from scurvy, and his sight was very much worse, but he could still see to read a little. At the age of 13 he ceased to be able to read. He now got occupation as a groom, and subsequently as a driver, being obliged to give up first one and then the other, as his sight gradually

failed. In 1841 he was at length obliged to give up all work, and has done none ever since. Up to two years ago he could just see to get about the street without using a stick. At present he has only the barest perception of a strong light. His aspect is that usually considered characteristic of amaurosis.

The irides are lustrous, the pupils larger than natural, but acting very fairly when exposed to the light.

It will be seen that the loss of sight has been very gradual indeed, its course extending over forty-one years.

He is not aware that any of his relations have had their sight affected, excepting a sister, who had her sight temporarily disturbed while working in scarlet cloth. His hearing is good, his smell is good, memory tolerable. There are no indications of general mischief to his nervous system. He is liable to pain across the eyebrows, but never in the eyes themselves. He has been a great smoker, but did not smoke before his eyes began to fail.

April, 1863. *Ophthalmoscopic Examination.*—The media are perfectly clear in both. The optic discs are of a dirty grey colour, and in both the vessels are reduced to the most extreme degree of minuteness. In each I can trace only one having an exceedingly small branch upwards and one downwards, probably veins. Although the choroids are very pale, their structure is not at any part absolutely wanting. There are no patches of exposed sclerotic. The branches of the Vasæ Vorticosæ in many parts are beautifully seen. The pigment is chiefly collected in the parts surrounding the optic disc and yellow spot, and becomes much less abundant at the periphery. Thus when he fixes the eye for inspection of the optic disc, at first sight the fundus appears almost black, so numerous are the pigment masses. Most of the latter are clearly in front of the choroid.

No. IX.—*A Child Born with one Eye deficient and the other shrunken.*

A baby, aged five weeks, was brought by her mother, Mrs. Hazlett, Oct. 11, 1866.

This was her sixth child, and the others were reported to be healthy. In the tenth week of her pregnancy she had a severe fright, being suddenly called into a room were a neighbour was dying. "The occurrence made her turn cold, and she did not

get warm again the whole day. The child was born at full time, but was very puny. All her others were fine ones." On examination we found that the child was exceedingly puny, and unable to make any noise in crying. There was some degree of talipes calcaneus of the right foot. It kept its fingers and thumbs constantly bent into the palms. Its head was of due size in proportion to its body. Its physiognomy was rendered peculiar by a deep furrow, which could be felt with the finger, crossing both orbits and the bridge of the nose from canthus to canthus. There was also a notch in the bony margin at the outer side of each orbit. The eyeballs were shrunken to a very small size. The palpebral apertures were very narrow, but the lids were perfect. The child shed tears when it cried. In the right orbit no trace of eyeball could be discovered. In the left orbit the bulb of the collapsed globe was distinguished by the remains of a small portion of cornea with a scar on it, and a tag of adherent iris looking exactly like the state of things after sloughing from purulent ophthalmia.

This child died a few weeks after these notes were taken. Dr. Gray, of Poplar, who made a post mortem, informs me that in the right orbit no trace of the eyeball could be found.

No. X.—*A Child Born with Shrunken Eyeballs.*

The subject of this case was shown me by my friend Mr. Mundie, of Richmond Road, who had attended the mother in her confinement.

The child presented a close parallel to what is described in the case above. The collapsed globes are so small, that they are with difficulty discovered. The child is living and healthy. Its head is well formed, and the furrow crossing the orbits described above is not present.

No. XI.—*On Excision of the Eyeball in the Acute Stage of Traumatic Pan-ophthalmitis.*

As an opinion is, I believe, held by some high authorities that the excision of globes when acutely inflamed is dangerous, I think it desirable to record the fact that during the last year I have performed numerous such

operations without the slightest ill result. Whenever I am satisfied that an injured globe is utterly lost, I always advise its excision without loss of time. By adopting this course the patient's suffering, often extreme, is at once put an end to, and I think, also, the risk of sympathetic inflammation of the other eye is avoided. I have excised globes in all stages of inflammation, and have never seen the slightest ill consequence, whilst the patients have invariably been most grateful for the complete relief afforded. The hæmorrhage is usully rather more than when the cellular tissue is not infiltrated, and the operation is much less easy of performance (especially when the globe is in a state of proptosis from inflammation), but these are objections of very minor consequence. In a recent case of peracute inflammation after a blow, in a poor girl of 18, the globe was pushed out of the orbit by swelling, the lids partially everted, and the conjunctiva chemosed. In this case, owing to the matting together of the tissues, and the soft, flabby state of the sclerotic, I had more difficulty in the excision than I could have believed possible. Although, after the removal of the globe, the lids were still left tense, dusky, and prominent from swelling, yet the most complete relief was given, and the patient, who had been worn out by ten days and nights' incessant suffering, was looking quite happy on the following day.

A month ago I excised an eyeball on account of traumatic ophthalmitis within forty-eight hours of the accident —by much the shortest period within which I have ever felt justified in advising the operation.* A lad of 14 was going through the street one Saturday evening, and stopping to watch a man shooting on a nut-stall, the cap flew, and a fragment entered his eye. Severe pain commenced at

* This statement, of course, applies only to removals on account of the inflammation, and not for the direct effects of the injury. Where the eyeball was hopelessly destroyed by laceration, I have sometimes removed its remains immediately.

once. He came to me on the Monday morning with a small hypopyon, the posterior lamina of the cornea dotted over with small accumulations of puro-lymph, acute iritis, and opaque vitreous. He had no perception of light. Mr. Dixon and Mr. Streatfeild both saw him with me, and both agreed in the opinion that, although the cornea was still almost clear, there could be no doubt that the organ was lost. The lad willingly consented to its removal, so great had been his suffering. After excision I found a portion of gun-cap behind the ciliary processes, the whole region of which was covered by puro-lymph in a coherent membrane. The vitreous was still, for the most part, translucent; but in various parts of its substance films of yellow lymph were visible. There was also lymph on the surface of the retina at almost all parts. At so early a stage of the inflammatory process, in such a structure, microscopic examination would have been most interesting, but I was compelled, by the pressure of engagements, to let the opportunity slip.

No. XII.—*Operations for Solution of Senile Cataracts commenced at an early period without allowing the Cataract to ripen.*

In a future number of our Journal I propose to give a detailed report of a series of cases in which I am now trying needle operations for senile cataracts. My plan is: 1st. To commence the treatment as soon as the opacity is advanced sufficiently to cause serious inconvenience to sight, and thus before the lens has become very hard; 2nd. To do very little each time, and to do that little very carefully, so as to avoid any displacement of the lens; 3rd. To allow a long interval between the operations; and 4th. To use atropine very thoroughly. In the cases now under treatment, eight in number, I have, as yet, had no drawback whatever. In all, solution is steadily proceeding. The patients have none of them been confined to the house, and

none have suffered any material pain. Even if in some it may prove needful to use a spoon to extract the nucleus, still I anticipate advantage from having got rid of the cortical substance beforehand.

No. XIII.—*Acute Glaucoma Supervening in an Eye previously lost by Retinitis from Renal Disease.*

Mrs. S., æt. 45, came to me suffering most severely from acute glaucoma in her left eye. The cornea was so steamy, and the media and lens so hazy, that it was impossible to inspect the fundus. The globe was very hard, and there was much venous congestion. As she told me that the eye had been blind for some weeks before the pain began, I had no hesitation in advising excision instead of iridectomy. In giving this advice I had also in view the possibility that a malignant growth might be present. She had scarcely slept for a week, and was worn out with pain. She had previously been under the care of another surgeon, who, after examining the eye, had requested a specimen of her urine, and by whom she had been sent to Hastings for the improvement of her general health.

I excised the globe, and found on dissecting it that the retina was greatly thickened by white fatty deposit in the region of the yellow spot, where also were numerous apoplexies. Her pain was completely relieved by the operation, and she soon regained her health. Her other eye is, as yet, perfect.

Her urine does not now contain albumen, but she has suffered from the kind of dyspeptic symptoms which are very common in cases of granular kidney.

No. XIV.—*Note on a Case of Double Glaucoma Six Years after the Attack.*

The following case illustrates the ultimate result of glaucoma under its two aspects: first, when let alone, and secondly, when arrested by operation. Facts as to the state which eyes that have been saved by iridectomy maintain many years afterwards are especially valuable.

Mrs. H., of Norwich, came under my care in July of the

present year on account of recurrent pain in the right eyeball. It had been lost by glaucoma six or seven years before, and presented now all the usual conditions. The globe was very hard, its surface dusky, and crossed by large veins; the ciliary region was much congested, and there was a dense secondary cataract. She had often suffered severe pain in and around it, and I advised extirpation as the only resource.

Her left eye had, she told me, been attacked by similar symptoms six years ago, and had been saved by a timely operation (iridectomy), performed by Mr. Critchett. At the date of this operation the right eye was already in so hopeless a condition that nothing was attempted on it. With her left eye she has ever since the operation enjoyed excellent sight, and she can now read small print easily for an hour together. Its tension and general aspect are normal.

I removed the lost eyeball, and afterwards made a hasty dissection of it. The vitreous was quite clear, and unusually firm. The iris was atrophied and very thin. The optic disc was deeply cupped and quite white. No trace of arteria or vena centralis was visible. Several small patches of extravasated blood were seen in the choroid, but there were none in the retina. I had not leisure to make a microscopic examination.

No. XV.—*Herpes Zoster of the Supra-orbital Nerve—Mild course, no inflammation of the Eye.*

I give the following case as an addition to my paper at page 194. It is the mildest case of frontal herpes that I have ever seen, though quite definite in character. The vesicles occurred only on the forehead, scalp and upper lid, only the supra-orbital territory being involved. The case supports my assertion, that unless the side of the nose shows vesicles the eye does not inflame.

Marianne Parker, æt. 14, strumous-looking, with thick lips, &c., was admitted November 19, 1866, with a scanty herpetic eruption on the left forehead, &c. Six days before she had felt pain in the left forehead and side of head. Three days later a rash had appeared on her forehead. There was then a small transverse group of herpetic vesicles on the upper eyelid, another on the inner part of the eyebrow, and a third on the side of the

head. There were none on the bridge or side or tip of the nose. Both eyelids were a little œdematous. There was no inflammation of the eye. The vesicles quickly dried up, but scars were left. A certain amount of irritability of the eye and intolerance of exposure to light attended the attack, and even remained for a time afterwards, but there was never any congestion whatever.

No. XVI.—*Shingles on the region of the left Frontal Nerve—No vesicles on the Nose, and no inflammation of the Eye.*

Mr. Paget has kindly sent to me the subjoined notes of another case of Frontal Zoster. They are from the pen of Mr. Ransome, of Bowden, Cheshire, by whom the patient was seen. We have in it another fact favouring the opinion that when the frontal region only is affected, the eye does not suffer materially.

A. B., æt. 43, head of a large firm in Manchester, generally enjoys good health, but subject to occasional attacks of dyspepsia, and ulcerated sore throat.

During last summer, after a period of hard work and anxiety, feeling languid and unfit for business, he was ordered to the seaside, whither he went at the beginning of September. Shortly after arriving there he experienced considerable "shooting" neuralgic pain over the left eyebrow, extending over the left side of the head. Some days afterwards several herpetic pustules appeared—one immediately over the supraorbital foramen—the others spreading in two chief lines, the inner one nearly vertical and extending nearly half way over the scalp, the other diverging across the left temporal region. One or two large pustules also appeared on the outer part of the left upper eyelid.

His medical attendant applied a strong solution of nitrate of silver and chloroform to the spots, and the following day severe erysipelatous inflammation set in and extended to the right side of the head, closing both eyes. There was also considerable suppuration from the spots. In a few days this subsided, no fresh pustules appeared, scabs formed over them, and gradually scaled off, leaving deep scars, like the pits from confluent small-pox. During and after the attack he was very weak and much depressed in spirits.

No. XVII.—*Inflammations of the Eye occurring some little time after Small-pox.*

Inflammations of the eye not unfrequently occur in patients who have suffered from small-pox, but who are quite convalescent before the eye is attacked. Mr. Marson, in his excellent report on this subject, of which I have given a summary at another page, makes but vague mention of this class of cases. They are indeed scarcely likely to come under the observation of the medical officers of the Small-pox Hospital, since their peculiarity is that they do not occur until the patient is well. At the Ophthalmic Hospital we not unfrequently admit cases of this kind. The eye having been attacked at a period varying from three to six weeks after the outbreak of the exanthem, a central ulcer of the cornea is the most common condition, and I have rarely seen both eyes affected, the ulcer frequently spreads rather widely, but not deeply. It is exceedingly intractable. There does not appear to me to be any good reason for alleging that these cases are merely scrofulous ophthalmia brought into action by the debilitating influence of a severe disease. Sometimes it occurs in delicate children, but half the cases I have seen were in adults of good health and who had never shown any indications of scrofula; nor could the keratitis be regarded as the direct result of debility, for the patients had regained appetite and a fair amount of strength, as proved by the fact that they were attending as out-patients, and in the habit of walking considerable distances. Tonics do not appear to assert any material good, or, at any rate, not quickly, and change of air has in one or two instances been the only influence under which benefit was obtained. A mild form of iritis also sometimes occurs as a sequela of small-pox. I have seen three or four instances of this, in two, at least, of which there was no inflammation of the cornea. I do not ever recollect to have seen both eyes affected.

Dr. Mackenzie mentions iritis as a not unfrequent combination of post-variolous keratitis when it occurs in adults; but he speaks of it as usually occurring about the twelfth day. That is, at the date at which the severe ulcerations of the cornea with hypopyon, &c., are usually observed. The cases to which I

refer were examples of almost pure iritis, and some of them were at much later periods.

No. XVIII.—*Notes on an Epidemic of Ophthalmia in a Pauper School.*

In November last I was requested by the Shoreditch Board of Guardians to visit their school at Brentwood, and report upon a form of ophthalmia which has prevailed more or less amongst the children for six years past.

In my visits I received every assistance from Mr. Butler, of Brentwood, the very able surgeon under whose medical charge the institution is. The school is a newly erected building in a very healthy situation, and well cared for in every respect. The diet of the children appeared to be good, and most of them bore the aspect of excellent health. The school contains 310 children, of whom about a third are under nine years of age. The dormitories, &c., are undoubtedly too crowded, and this necessitates the opening of windows and the risk of draughts.

Mr. Butler informed me that when the school came to Brentwood, in 1854, some of the children were suffering from ophthalmia, and that they had never been rid of the disease since. Six years ago a very severe epidemic prevailed. During the last four years it has been comparatively limited until the present summer, when it began to prevail about the middle of May. It had usually been worse during the summer and winter months. When epidemics had occurred they had almost all commenced suddenly. The boys had usually suffered more than the girls. During the present summer many of the "infants" (under seven years of age) had had it. I examined all the children, of whom about a third had inflamed eyes. The inflammation was of the chronic catarrhal form. In a few cases the lids were granular, but in none could I detect any evidence of enlargement of conjunctival glands ("sago-grains"). In a very few cases, probably not connected with this epidemic, ulcerations on the cornea had occurred. In no instance that came under my notice had the disease passed into a severe type of purulent ophthalmia. In no case had the eye been lost, nor did there appear to be any danger of material damage in any under treatment.

It appeared clear to me that the ophthalmia was a mild form

of contagious or catarrhal conjunctivitis, and I inclined to attribute its spread entirely to contagion. As compared with large numbers of the children of the poor living in their own homes these children had certainly great advantages both as regards diet, clothing, and ventilation.

The only peculiarity of which I could take hold, was the fact that so large a number were brought under one roof, and hence the facilities for the spreading of a contagious malady. A single child coming new to the school suffering from catarrhal ophthalmia might become the focus for the infection of most of his companions. Mr. Butler agreed with me in the belief that this had frequently occurred, and said that it was not uncommon for children to be sent up from London while suffering from sore eyes. The fact that the ophthalmia had prevailed sometimes in one part of the school, sometimes in another (boys, girls, and infants), favoured the belief that the disease spread by contagion, and contagion only.

Acting upon this opinion as to the real nature of the disease, I strongly recommended that increased hospital accommodation should be obtained so as to allow of the complete isolation of those affected until quite recovered. This seemed to be the chief measure. In addition to it we of course continued the local measures of treatment, which had hitherto been found very fairly successful, suggesting only some very slight alterations as regards the strength of the solutions used.

1. A CASE OF EPITHELIAL CANCER OF THE ORBIT.

By J. W. Hulke.

The deceptive resemblance of the external swelling to that of a carbuncle, the rapid growth of the cancer, its close sequence on a violent blow, and the safety with which the chloride of zinc paste was used for the purpose of destroying any portions of the morbid tissue which might have escaped removal by cutting, make the following case of orbital tumour one of the most instructive and interesting which I have seen:—

A railway porter, æt. 42, struck his left cheek against the buffer of a carriage, and three days afterwards it became black as if bruised. Three weeks after this the cheek swelled, and the eye began to water, and in two or three days more the swelling spread to the eyelids. About this time there was bleeding from the left nostril, which has occurred occasionally ever since.

February 14th. Six weeks after the date of the injury he became an out-patient at the Royal London Ophthalmic Hospital, under my colleague, Mr. Wordsworth. At that time the lower eyelid and the skin at the inner canthus were distended, red and shining, and the eyeball was slightly displaced forwards, and to the temporal side of the orbit. Mr. Wordsworth, thinking there was an abscess, made an incision, but no pus escaped from it; and a week later he again deeply punctured the swelling with a narrow knife, with a similar negative result.

A few days after this I first saw the patient. Now the lower eyelid, especially at its centre and inner end, was very much swollen; the hollow between the eye and the nose was filled up by a prominent dusky-red swelling, dotted with small yellowish points, which had the appearance of pus, but when cut were found to contain a soft gelatinous substance. The eyeball was thrown considerably forwards, upwards, and to the temporal side, and its movements were much restricted.

The conjunctiva was red and œdematous, and filmed over with mucus. The caruncle, in the form of a large flattened tumour, protruded between the eyelids. The cornea was bright. V. = No. 19, J. at 8″. The swelling and hardness of the tissues ceased with a definite margin above, but below, shaded off imperceptibly into the cheek. A director passed into the second incision ran with very slight resistance, as if through a soft tissue, along the inner wall to the apex of the orbit, and on withdrawing it the groove was found filled with a soft gelatinous substance, which was not more minutely examined, because no microscope was at hand. At 2½″ bare rough bone was felt.

The left nostril was much obstructed, which, he said, had been the case for a fortnight. This appeared to be partly the result of an old injury, which, eight years before, had permanently bent his nose to the right, but mainly to proceed from a general puffiness of the mucous membrane, and from an adventitious growth, apparently a simple gelatinous polypus.

Two of our colleagues regarded the swelling as carbuncular cellulitis, and its close sequence on the blow seemed consistent with the opinion that it was inflammatory; but the characters of the swelling of the lids, its colour and consistence, more particularly the sago-grain-like dots in the skin, and the great enlargement of the caruncle, reminded me so forcibly of what we sometimes see when the glands and skin under the jaw are rapidly infected in those exceptionally rapid cancers of the tongue which run their course in a few weeks, that I could not come to any other conclusion than that the disease in the present case was cancerous. It was evident, from the proptosis, epiphora, and epistaxis, that the morbid growth had already extended into the orbit behind the eyeball, and now also invaded the nostril, and that surgical interference, to be of any use, should be early.

Knowing my interest in orbital tumours, Mr. Wordsworth courteously transferred the patient to my charge, and, March 2nd, I took him into the Middlesex Hospital, in order that I might watch him more closely than I could at Moorfields.

At a fresh consultation (at the Middlesex Hospital) most of my colleagues concurred in the view taken by two of the Moorfields Hospital staff, that the disease was inflammation of the cellular tissue, but one coincided with me that it was cancerous, It was agreed to watch the case for a few days longer.

I mention these facts to show the difficulty of forming a positive diagnosis.

Compresses soaked in goulard water were applied, and quinine gr. ii. were ordered to be taken twice a day.

March 6th. Since the last date there has been bleeding several times from an ulcerated spot in the caruncle; the first time it was only a few drops, but subsequently it increased to a continuous trickle. He was easier after the bleedings. He had now a worn, anxious expression; his nights were disturbed by a hacking cough, for which ether and henbane were prescribed.

March 9th. No more bleeding; his cough harasses him, causing much pain in the brow. The caruncle and lower eyelid are more swollen, and the hardness and redness have spread further in the cheek. An increased number of sago-like grains are now visible in the lower lid.

13th. The swelling is greater; he has more pain; a slight fulness is perceptible in front of the tempero-maxillary joint, and behind the angle of the mandible. Are these enlarging lymphatic glands? It had now become evident that complete extirpation of all the diseased parts afforded the only chance of prolonging, if not saving life, and the rapid rate of growth made it equally plain that further delay was not justifiable. The patient, when candidly informed of his desperate condition and prospects, accepted the slight chance held out to him by surgical interference.

14th. Having put him under the influence of chloroform, I made a preliminary incision into the tumour, and a small piece having been taken from its interior, the microscope shewed it to consist mainly of large mono- and poly-nucleated cells of an epithelioid type, which could not possibly be mistaken for any inflammatory product.

The diagnosis of cancer being thus positively confirmed, the eyeball was first enucleated (although not actually implicated, this was necessary in order to get at the intra-orbital part of the tumour behind it); the close adhesion of the tumour to the ocular capsule (O'Ferral's) occasioned a slight difficulty in doing this. The eyelids, and all the brawny part of the cheek and of the side of the nose, were encircled by a cut down to the bone, and rapidly dissected away. This done, I found the whole orbit filled with a soft, friable, succulent mass, which passed through

a gap in the orbital floor, and nearly filled the antrum, without, however, having any connection with the walls of this latter cavity, the surfaces of which were lined by normal mucous membrane. This cavity also contained a little glairy mucus.

The tumour had also extended into the nose through the osplanum, and filling the back of the left nasal passage, projected in the form of a capsulated globular lobule through the posterior nares into the pharynx above the soft palate. (A soft simple polypus was taken from the front of this nostril.) In order to follow the tumour in this direction, I had to cut away much of the maxilla, the left nasal bone, and the lateral mass of the ethmoid. It was now possible to detach the periosteum from the bones with a raspatory wherever the tumour appeared to have been connected with these latter, and when this had been done the bony surfaces were rubbed over with a hot iron. Strips of lint, spread with chloride of zinc paste, were applied to the entire wounded surface of the bone and soft parts. This completed the operation.

The bleeding was very free, but yet less than I had anticipated, and had quite ceased before the zinc was applied. The enlarged ophthalmic artery cut at the apex of the orbit continued to bleed in spite of the cautery, but was stopped by a small compress dipped in Liq. ferri perchlor.

The frontal sinus was opened in the course of the operation, and found not to be involved.

The limits of the tumour were much more extensive than I had anticipated, particularly towards the pharynx and the antrum, for the body of the maxilla was not expanded nor the palate depressed.

The minute structure was characteristic of epithelial cancer. The preponderating histological elements were large scale-like epithelial cells, closely packed in cylindrical masses and in concentric clusters. These, with a small quantity of fibrous, interstitial connective tissue, and blood-vessels, made up the great mass of the tumour in which no remains of the normal tissues were found; but along the line of extension where cancer was invading the normal tissues, there was a thin stratum of a new cell-tissue

pushing out organoid buds among the normal tissues, and this new tissue consisted of small roundish cells about the size of those of the deeper layers of the rete mucosum of the cutis.

For some hours after the operation, I was anxious lest the fluids from the wound, saturated with chloride of zinc, trickling over the upper surface of the velum palati into the throat, might excite inflammatory œdema of the fauces and epiglottis, or perhaps poison the patient, but nothing of the kind occurred.

15th. Pulse 104; skin cool; tongue clean; slight swelling and redness of the edges of the wound and slight œdema of the right eyelids. His diet was strong beef-tea and a couple of eggs, and brandy ʒviii, but he said he had not enough to eat, and asked for a mutton chop, which was given him.

16th. Pulse 100; slept fairly; slight extension of the inflammatory redness. Complains of headache, which was relieved by a caoutchouc bag of ice. It would be too tedious to detail the daily report, and from this time I shall only mention the leading points. The dry, hard bark-like eschar formed by the caustic zinc was soon detached at its circumference by a narrow line of ulceration, which was quickly followed by cicatrization and shrinking, so that when the entire eschar fell way, April 11th, the wound was much smaller than at the time of the operation.

April 24th. At the upper and inner angle of the orbit, and on the septum nasi, the granulations are rather exuberant. Does this overgrowth arise from the irritation of small sequestra not yet detached, or does it mean a recurrence of the cancer?

Several sequestra were withdrawn from among the granulations, which subsequently assumed a more healthy appearance.

May 7th. The outer edge of the orbit is becoming loose.

23rd. A large hard fixed nodule has become palpable since the last note, behind the angle of the jaw. Three days later this had much enlarged.

29th. The loose edge of the orbit was lifted off.

June 1st. A fleshy knob has sprouted from the lower and outer part of the orbit corresponding to the spheno-maxillary

fissure. The manifestly cancerous infection of the lymphatic gland, noticed on the 23rd, made it useless to destroy this. Both it and the gland behind the jaw increased, so that by June 8th he could not chew solid food, and could only separate the jaws to a very slight extent.

29th. Deglutition is now difficult. The tumour behind the jaw is very prominent, and its summit is soft and red; it is evidently about to break. The orbit is becoming filled with a growth from the outer wall.

In a few days the tumour behind the jaw broke and fungated, and from this time bled frequently. It widely infiltrated the neck, and from time to time large portions sloughed away, meanwhile the orbital growth made little progress. In spite of the frequent loss of blood and the profuse discharge from the wound, he survived till November 29, 1866.

Mr. Arnott, our Surgical Registrar, has supplied me with the following memorandum of the post-mortem examination:—

"There is a large sloughy cavity in the place of the left orbit which reaches deeply towards the pharynx, and outwards to the zygoma, while at its inner side are seen the remains of the septum narium. A protuberant, fungating ulcer reaches from the edge of this cavity to below the lower jaw. It involves all that remains of the cheek, and below it nearly as far as the collar-bone, the neck from the middle line, below the ear, nearly to the vertebral spines, is hard and swollen. The right side of the neck, below and behind the jaw, is similarly swollen in a less degree. The swelling proceeds from a cancerous growth which has destroyed the outer bony wall of the orbit, the part of the malar bone not removed at the operation, a portion of the ascending ramus of the lower jaw, and all the soft tissues in these situations. The sterno-cleido mastoideus muscle is lost in the centre of the cancerous mass, which pushes aside but does not structurally implicate the arteria carotis communis or vena jugularis interna. There were a few scattered knots of chalky tubercle in the lungs, evidently of old date, and with this exception the thoracic and abdominal viscera were healthy. No secondary infection of distant lymphatic glands was found."

In the progressive, continuous infection of the neck, and the absence of distant, interrupted infection, we see the

same features which are so generally observed in epithelioma of the lip and uterus, which, while they infect the next lymphatic glands, are rarely accompanied by secondary tumours in distant organs. It is just this minor diffusibility which makes epithelioma less desperate than scirrhus, and brings it more within the scope of surgery. The longer average duration of life in epithelioma over that in other forms of cancer, 53 months according to Sibley, proceeds from the same cause. This case, lasting only about 10 months, was therefore an unusually rapid one, which makes the postponement of the operation the more to be regretted.

Although operative interference in cancer of the eyeball or orbit is discountenanced as useless, or worse than useless, by some surgeons, these constitute a very small and rapidly decreasing minority, and it is generally allowed that thorough extirpation alone offers a prospect of immunity from recurrence, relative or complete, whether the tumour be a carcinoma *stricte sic dictum*, or an infecting sarcoma. But here unanimity ceases, for beyond agreeing that thorough extirpation is only to be hoped for at an early stage, when the growth originates in the bones or in the periorbita, we find surgeons of the highest reputation not agreed as to the best way of operating, but practising different methods, some of which, from their very nature, involve the practical non-observance of that principle on which every operative measure should rest, viz., that it should be thorough for the particular end in view. Perhaps the chief reason of this is the supposition that the situation of the orbit, especially its nearness to the brain, does not admit of the same methods of operating as are advantageously employed upon the trunk and limbs; while a minor reason is the want of personal familiarity with the resources of general surgery by those who exclusively devote themselves to a special branch.

As far as I have seen and read, the two methods of removing orbital tumours in common use with us are exci-

sion of the tumour with knife or scissors, and its evulsion with blunt instruments. The first I have, till lately, always adopted myself, too often to see before long the tumour sprout up anew. Nor is this surprising, since diffusibility, infectiousness, is the leading feature of carcinoma, and in a scarcely less degree of some sarcomata; and the larger number of orbital tumours fall into one of these classes. The microscopic examination of the tissues around a cancerous breast, at points where neither touch nor unaided eye could detect any traces of the morbid growth, has again and again proved their already infection; and in an intraocular cancer, where the sclerotic appeared, on close scrutiny, to be entire, I have found the loose areolar tissue outside the eyeball, between the sclerotic coat and the capsular fascia, as well as that between the outer and the inner sheath of the optic nerve, to be microscopically infiltrated. It is therefore evident that the removal of those tissues only which are so grossly diseased as to be appreciable as such by sight or touch is insufficient, and that we ought to adapt to the orbit the maxim which enjoins the excision of a zone of normal tissue whenever we excise a cancerous or other infecting tumour seated in the trunk.

The same incompleteness inseparable in most instances from simple excision, belongs in a much greater degree to the practice of tearing out orbital tumours with blunt elevators. Cysts and encapsuled tumours may no doubt be entirely enucleated with these instruments, but an infiltrating growth, such as cancer, which has no distinct definite boundary, cannot be cleanly shelled out with them. Whenever I have seen them used the tumour has been torn in pieces, and so ragged a wound has been left that a suspicion, sometimes amounting to a conviction, that shreds of the morbid growth have been left has forced itself upon me. But if these blunt instruments are unsuitable for the complete extirpation of an infiltrating tumour, there is also an additional reason why they should not be used for

this purpose. It requires so much force to wrench away the tumour from the orbital walls, that these latter may be inadvertently pierced, and the elevator or director plunged into the brain; or pieces of the roof of the orbit may be torn away without the operator being at the moment aware of the damage he is inflicting: both these accidents have actually happened.

The best method is, I believe, a combination of excision and escharotics. By clipping out the mass with a blunt-pointed scissors, curved on the flat, strong but not unmanageably large, and carefully exploring with the finger as we proceed, we avoid the risks incurred when elevators are employed. In this way I have safely removed both very tough and very friable tumours. When the orbit has been thus emptied, if its walls are anywhere compromised by the disease, the periorbita can be detached with an elevator or a raspatory, and as much of the bone itself as is necessary removed, and this safely, because the part acted on is directly under the finger, and the degree of force used can be exactly estimated, and its direction controlled. This done, for the double object of stopping any bleeding vessels, and completing the destruction of any part of the tumour inaccessible to the scissors, the wounded surface should be touched with the hot iron, and when the bleeding has completely ceased, the operation is finished by coating the wound with lint spread with the escharotic zinc paste, the formula for which I gave in the last number.

The advantages of this escharotic are its manageability, the small amount of local inflammation, and the slight symptomatic disturbance which follows its use, even when it is applied to a very large surface. The depth to which it will bite can be regulated with some certainty by the thickness of the layer of the paste, and the eschar is a hard dry bark, the separation of which is followed step by step by cicatrization, so that by the time it is fully detached the wound is not unfrequently reduced to one-third or less

of its original size. These characteristics of the zinc escharotic allow us to apply it to the orbital walls with more safety than any one who had not watched its use would be inclined to credit. Of this the case to which these remarks are appended is a good example. The practice was originated, as far as I know, in the Middlesex Hospital by Mr. Moore, and it has been adopted by Mr. De Morgan, Mr. Lawson, and by myself, in several cases with very encouraging results. In no instance has fatal intracranial inflammation been provoked, but I may not conceal that in three instances there was considerable excitation manifested by epileptiform fits a few hours after the operation. These, though sufficiently alarming, ceased, and did not recur. There is a disadvantage about which I must add one word. During cicatrization the eyelids are drawn into the orbit, so that an artificial eye cannot be adopted. No doubt this is a drawback, but one of little moment, seeing that the stake is life.

2. A CASE OF ORBITAL SARCOMA FOR COMPLETING THE EXTIRPATION OF WHICH THE CAUSTIC ZINC-PASTE WAS USED WITHOUT INJURY TO THE EYEBALL.

By J. W. Hulke.

In the case just related, and in that which I recorded in the last number, the zinc paste was extensively applied after the removal of the eyeball. The following shows that, with precaution, it may be used in suitable cases, where the eyeball is present, without injury to this latter:—

A girl, æt. 18, came to the Royal London Ophthalmic Hospital June 6, 1866, with a swelling in the situation of the lachrymal sac. She said that five weeks before, after exposure to cold, the right side of her face had become red, swollen, and shining, and that for two years previously she had been troubled with epiphora.

The history was that of cystitis following obstruction of the nasal duct, and the swelling, at a cursory glance, was not unlike a distended lachrymal sac. It was, however, incompressible, and its contents could neither be squeezed away into the nose nor did they regurgitate through the lachrymal puncta. This led me to examine it more carefully. It was evidently closely connected with the inner wall of the orbit. Its surface was even, and its outline oblong. Its upper border, above the tendo oculi, was indefinite, and its lower end, in the form of a free, round knob, reached to the junction of the inner and middle third of the lower margin of the orbit. The swelling was elastic, but harder than most cysts. The skin did not adhere to it, and there was not any œdema of the neighbouring tissues.

From the solid feel and lobular form of the swelling, I suspected a new growth; but two of my colleagues were disposed to regard it as a mococele of the lachrymal sac. The Royal London Ophthalmic Hospital being full, I sent the patient into the Middlesex Hospital, placed her under chloroform, and to put the question of sac beyond doubt, slit up the lower punctum and canaliculus into the sac, and then passed a large probe through the nasal duct into the nose. The passage was pervious, no pus or mucus escaped, and the size of the swelling was not changed. The diagnosis being thus confirmed, I dissected out the tumour which sprang from the periorbita behind the sac, sparing the conjunctiva which it had not implicated, when the slight bleeding had quite ceased, and coated the nasal side of the wound, where the base of the tumour had been attached, with the chloride of zinc paste spread on small pieces of lint, the dry surfaces of which were turned towards the conjunctiva which was further protected against the caustic by a little charpie. The conjunctiva and eyeball with its muscles were not injured by the caustic, the eschar exfoliated, the wound healed soundly, and during the time the patient remained under observation there was no sign of recurrence. The histological characters of the tumour were those of a spindle-cell sarcoma.

REPORT ON CASES OF CONGENITAL AMAUROSIS.

By Jonathan Hutchinson, F.R.C.S.

I publish the following cases as a contribution to our knowledge of the conditions under which congenital blindness occurs. They are all in which I possess notes of ophthalmoscopic examination. My comments on them may be arranged conveniently under the following heads:—

Marriages of Consanguinity.—In but one instance were the patients the offspring of marriages of relations (Cases 2 and 3). In this case, however, two children had suffered. Neither of them were born absolutely blind, but both were nearly so, and both had white atrophic optic nerves. Both were intelligent, and enjoyed the full use of the other senses. Although in several of the other cases no note as to parents' history is recorded, yet this absence is to be held conclusive as to the point referred to, as I never omit to inquire.

Microcephalism and Idiotcy.—Two of the cases were examples of microcephalism in an extreme degree, and the children were probably idiots. In one of these the discs were white, whilst in the other, although the blindness was total, no ocular changes could be detected. In a third case the patient was microcephalic in moderate degree, and the optic discs were white and the choroids extensively absorbed. In all these early closure of the fontanelles was observed. I may here add, that during the past few years I have seen several extreme instances of congenital microcephalism in which the function of sight was yet perfect.

Hydrocephalus.—In only one instance was there any tendency to hydrocephalic effusion (Case V). In this instance the discs were grey white.

State of Pupils, &c.—In all the cases the irides were brilliant and responded well to atropine. In most of them

the pupils were of normal size and sluggish, but in several, although the child was blind, they acted moderately. In most cases a certain amount of oscillation and twitching of the globes was observed.

The Optic Discs and Vessels.—In one case no change in the disc or its vessels could be appreciated, the child yet being microcephalic and quite blind. In one, after two patient examinations, no disc nor any trace of a retinal vessel could be found. Of the six others, in all the disc was atrophied, grey white, and depressed, and in most the atrophic changes were far advanced. In Cases II and III, the retinal vessels were extremely shrunken ; in Case IV they were small, whilst in the others their diminution in size was but slightly marked.

The Choroid.—In Cases I, II, III, V, VI, and VII, the choroid showed no morbid changes, whilst in Cases IV and VIII there were evidences of past choroiditis in the presence of large patches where that membrane was wholly absorbed and the sclerotic exposed.

Conjectures as to Cause.—Intra-uterine disease (neuritis, &c.) suggests itself as the most probable cause in a majority of the cases, those, namely, in which the optic disc was atrophied, or in which the choroid was absorbed. The present condition of some of the patients closely resembles that of some of those recorded in my paper on Neuritis in Children, at page 307 of the present number. If we suppose that the infants had, during fœtal existence, suffered from intra-cranial disease of similar character to that which the other children had shown at a later period of life, we should have a good explanation of the conditions now displayed. The subject is a very interesting one, and I hope at some future period to record a more detailed and extensive series of cases.

A Tabular Statement of Eight Cases of Congenital Amaurosis.

No.	Name. Reference to note-book. Date of notes.	Age.	Development of head and state of health.	State of Intelligence and of Special Senses.	History of Parents, &c., &c.	State of the Pupils, Eyeballs, &c.	Ophthalmoscopic Examination.	Remarks.
1	Mary Anne Raynham, p. 13, B. October 14, 1861.	4	Born healthy, began to be "ailing" at 10 months. Is small for her age, but well fleshed and florid; in good health for previous year. When two years old she had no use in her ankles and could not walk.	Her mother doubted whether she could see a candle. In the daylight could not see the largest objects. She walked steadily. Backward in talking, but could now say almost anything.	Her mother stated that during pregnancy she was suffering from great anxiety. She believed the child to have been born blind.	Both pupils large, but not remarkably so; equal and round. Irides blue, lustrous. Globes of good size. The eyes looked well enough, but the globes twitched.	Very difficult to examine with the ophthalmoscope, owing to the motion of the globes. Media quite clear; choroids florid; retinal arteries small; veins of full size. Optic discs blue white (but not so conspicuously so as is often seen), and depressed.	
2	Mary Anne Venables.	4	Both quite healthy since birth. The eldest did not walk till two years old, and the youngest	Quite intelligent, clever children, hearing perfect, good tempered. The eldest only saw enough to avoid large objects; would often run against a	Father and mother first cousins. No blindness in the family; no insanity. A boy was born, but he	Fair complexion; grey eyes. In the elder, pupils of normal size, and acted fairly. The globes	*The elder one.*—Both optic discs perfectly white, and their vessels shrunk to the most extreme degree. Neither arteria nor vena centralis to be	These children were the offspring of a marriage of consanguinity.

A TABULAR STATEMENT OF EIGHT CASES OF CONGENITAL AMAUROSIS—*(continued)*.

No.	Name. Reference to note-book. Date of notes.	Age.	Development of head and state of health.	State of Intelligence and of Special Senses.	History of Parents, &c., &c.	State of the Pupils, Eyeballs, &c.	Ophthalmoscopic Examination.	Remarks.
			could now just stand.	chair. The younger saw the clock. Her mother thought her getting worse, and believed that she could see well till six months old.	lived only four months. His sight was believed to be good. No other children.	oscillated somewhat in both.	traced beyond the margins of the disc, and they could only just be discovered on the disc itself.	The elder one was an instance of congenital blindness, the younger was believed to have enjoyed some sight for 6 months. Neither of them were as yet absolutely blind.
3	Emily Venables, p. 63, B. August 14, 1862. Sister of the above.	1½					*In the younger.*—Great difficulty in examining the fundus. Optic discs large, and very white, bluish white and dead, without a trace of pink. The arteria and vena centralis very small, but not nearly so small as in her sister.	
4	Margaret Louisa Flitt, p. 158, B. January 8, 1866.	10 wks.	Intelligent and healthy, but its forehead and upper part of head	It grasped with its hands readily. Appetite large, never satisfied. Too young to test the other	The parents were not related before marriage. No history of	The globes twitched a little. The pupils dilated well	There were very large abrupt patches of absorbed choroid where the sclerotic was quite bare.	This was an eight months' child. A case of

			decidedly small, and the anterior fontanelle already all but closed.	senses.	blindness in the family. An elder sister eighteen months old quite healthy and enjoying good sight.	with atropine, but owing to the child's struggles it was difficult to get a sight of the fundus.	There were not many large accumulations of pigment. I did not get a good view of the disc itself in either eye, but I believe that it was in each quite white, and the vessels small.	microcephalism, though not to an extreme degree.
5	Alice Maud Rose, p. 232, B. February 12, 1866.	3 mo.	Head large and fontanelles very widely open. There was a superficial nævus on each upper eyelid.	Features well formed. Hearing good.	The mother's first child.	Pupils of normal size. She rolled the eyes about, but the globes did not twitch.	Discs grey-white, with black rims, the margins being distinct. Veins rather large, the arteries rather small. There was no evidence of neuritis nor retinitis.	
6	Mary Ellwood, p. 258 B., May 8, 1865.	15 mo.	Forehead small, face and ears large. Fontanelles early closed. The mother stated that they had always been so.	She did not seem to notice anything brought before her, but she noticed the slightest noise. *Dec. 11th.*—The child appeared idiotic, never took hold of anything. Her limbs were flabby and thin, and she made no attempt at standing.	The fifth child, one died from measles. The eldest was nine years old. They were all fine, healthy children. The parents were not related before marriage.	Atropine dilated the pupils well.	We administered chloroform Dec. 11, and examined carefully. Nothing whatever abnormal could be detected. The discs were of good colour, and the vessels of good size.	A case of microcephalic idiotcy. The absence of optic nerve disease is important.

A TABULAR STATEMENT OF EIGHT CASES OF CONGENITAL AMAUROSIS—*(continued)*.

No.	Name. Reference to note-book. Date of notes.	Age.	Development of head and state of health.	State of Intelligence and of Special Senses.	History of Parents, &c., &c.	State of the Pupils, Eyeballs, &c.	Ophthalmoscopic Examination.	Remarks.
7	Joseph Lane, July 12, 1866, p. 46, C.	10 wks.	Head a little too narrow in front and too broad behind. The whole head rather small.	Note omitted.	Note omitted.	Note omitted.	Both discs grey, their margins indistinct. Choroid and retina healthy. The central artery of medium size, but somewhat diminished.	
8	An Infant. Nov. 27, 1863.	6 mo.	Head very small; forehead low, and very narrow.	Expressionless physiognomy. Could, however, be made to smile occasionally, and would take notice when spoken to. No perception of light.	Note omitted.	Pupils normal, and dilated well with atropine.	The optic discs could not be found, nor were any retinal vessels seen. Large patches of choroid were absorbed, exposing the sclerotic, which at these parts was not crossed by a single vessel.	A case of microcephalic idiotcy. It is possible that the optic nerves were wanting, but the examinatien was made under difficulties.

CASES SELECTED FROM MR. DIXON'S OUT-PATIENTS.

Complete paralysis of the left fifth nerve as regards sensation—Motor function unimpaired—Lachrymal gland active—Impaired taste and smell—Superficial ulceration of the cornea—Suspicious symptoms as regards syphilis.

(Reported by Mr. W. Square, Clinical Assistant to Mr. Dixon.)

Charlotte Waters, æt. 30. was admitted on December 3rd, 1866, a healthy-looking woman; a widow for ten months. Has had three children, of whom two are alive and one died in teething. The survivors are perfectly healthy. Her husband was "a healthy man, but subject to abscesses in various parts of his body." She has had a diseased upper jaw for four years on the left side. There is a scar at the ala of the nose, where bone has been extracted. She has lost four teeth on that side, three molars and a bicuspid. They simply dropped out, without any pain or swelling, and were not decayed. The rest are good. There are two small sores at the inner canthus, one above and the other below the caruncle. They were made by nitric acid to cure small ulcers. The vessels of that side are slightly engorged. She applies to this hospital on account of ulceration of the cornea. It is extensively and superficially abraded. This began fourteen days ago. The conjunctival vessels are injected. The iris acts well. There is total loss of sensation over all parts supplied by the fifth nerve on this side. All the skin of the face with the exception of the part supplied by the cervical plexus, viz., between the meatus of the ear and the angle of the mouth, is without sensation. The cornea and conjunctiva are in the same condition. The loss of sensation extends into the mouth, implicating the hard palate, soft palate, tongue, gums, and inside of the cheek. She has no smell or taste on that side. The hearing is good. The lacrymal gland acts, as she has noticed that she cries with both eyes. The motor root does not seem to be implicated. The masseter is of fair size and acts well. The left temporal muscle, perhaps, is a trifle smaller than the other. She cannot count fingers with

the left eye, but has perception of light. She says that the sight was quite good until the cornea began to ulcerate. It is too dim to admit of an ophthalmoscopic examination. There has never been any pain. The upper maxilla and the malar bone are tender on firm pressure. There is apparently more necrosed bone to come away.

There is no positive evidence in this case to support the diagnosis of syphilis. The character of the ulceration in the cheek is very suspicious, and is rendered more so by the fact that the alveolus is diseased.

Partial paralysis of the left fifth nerve, both of its motor and sensory functions—History of Epileptic fits and of pain in the parts supplied by the nerve now paralysed—Impaired taste and smell—Ulceration of the cornea—Doubtful diagnosis of syphilis.

(Reported by Mr. W. Square, Clinical Assistant to Mr. Dixon.)

Sarah Stone, æt. 27, admitted December 6, 1866. A pale, unhealthy-looking woman, subject to fainting fits. She has had rheumatic fever and has pains constantly flying about her limbs. She is very subject to sore throat and a rash on the skin with indurations under the spots. She believes that she inherits epilepsy from her father. Her husband is a soldier and in good health. There is no confession of syphilis on either side. She has had iritis in the right eye, which has left adhesions at the lower and outer part. She, however, reads brilliant with it. About four months ago she had three epileptic fits, which were followed by pain all over the distribution of the fifth nerve of the left half of face. She found, too, that there was diminished sensibility in that side. The pain was worst in the eye, especially at night. The cornea has a leucoma as from old keratitis. She can only discern light with this eye. The conjunctiva is slightly congested. It is sensitive but not nearly so much as the other. The under lid has entirely lost its sensibility. In the rest of the skin supplied by the fifth sensibility is only impaired, not lost. Taste and smell are both defective, and she complains of a nasty taste at times. The masseter and temporal muscles act, but not very well, and she complains of loss of power on that side. The

motor seems to be implicated to the same extent as the sensory root. She has had enlarged cervical glands. Her family is reported to be generally healthy.

In this instance the suspicion of a syphilitic taint is strong, although there is no conclusive history. The woman is liable to sore throat and to rash, and has had a former attack of iritis.

Blindness with white atrophy in one Eye, with paralysis of third and fourth nerves on the same side—Occasional Hemiopia and failure of sight in the other Eye.

(Reported by Mr. WAREN TAY, Clinical Assistant to Mr. Dixon.)

In the following case the symptoms seem to point clearly to the existence of a new growth at the base of the brain already involving most of the orbital nerves on one side, and slowly extending to the other.

Mrs. Manifold, aged 27, came under Mr. Dixon's care in the autumn of 1864. The following is the note made at the time:—

She is tall and thin, and does not look specially out of health. She has been married for seven years, and her husband is in the 1st Life Guards.

She thinks that her right eye began to fail twenty months ago, and that it has been quite blind for nine months. There was no drooping of the lid at first, only during the last two months, and it has often passed off for a time. She has had very severe pain in the head at times, attended by a very peculiar excitable feeling, as if she should go out of her mind. She had a blow on the right eye six years ago, but has never had any fall or severe injury to the head. Once, for about a week, she thought her right arm was becoming paralysed, but it passed off. She is very cheerful, and remarkably voluble in speech. There is no history of symptoms suspicious of syphilis, and when questioned directly no clue was obtained. She has been much under treatment for her eye, and the pain in her head, but with no material relief. She has often been giddy, but has never actually fallen down. She has never been sick. She can walk well, and con-

siders herself stronger now than she was formerly, and she has much less pain in her head now than some months ago.

The right eyelid drops so as completely to cover the eye, and she has not the slightest power of lifting it. The eye is strongly everted, and the pupil dilated to thrice its normal size and fixed. She has no perception of light. She cannot move the eye upwards or downwards. The fourth nerve appears also to be quite paralysed. The sixth acts strongly. The left pupil is large and sluggish, but will act when exposed to light. She can with this eye with difficulty spell letters of No. 14 (Jaeger). She complains that after looking stedfastly, a black shade will cross her sight. Sometimes she can see only the half of an object. There is no drooping of the eyelid on this side. On examination with the ophthalmoscope the optic disc of the right eye was found white and atrophied; that of the left paler than natural, but not to any great degree.

December 11, 1865.—Mr. Dixon states that she is gone to Windsor with her husband, and that when he last saw her she was in the same condition as that above described.

It is somewhat unusual that no stage of severe head symptoms should ever have occurred in this case. The changes in the nerve appear to have been those of atrophy without neuritis. The circumstance that one eye should escape for so long an interval is also decidedly exceptional to what we usually see in this group of cases.

PART II.

A Periscope

OF

CONTEMPORARY OPHTHALMIC LITERATURE.

PROFESSOR VON GRAEFE ON EXTRACTION OF CATARACT.

IN the Archiv. f. Ophthalmologie, vol. 12, part 1, Professor v. Graefe has published some lengthy supplementary remarks upon his now well known method of "modified linear extraction" of cataract. Speaking from a personal experience of about 300 operations, the Professor states that in 90 per cent. of them he has obtained a complete result, with acuity of vision between $\frac{1}{6}$ and $\frac{5}{6}$. Of these cases, in 82 per cent. the healing process was absolutely normal, and in the other, 8 per cent. was interrupted by some transitory accident. In 10 per cent. of all the cases operated upon the results were unsatisfactory; but among these the majority were cases of imperfect success, admitting of improvement by subsequent operation. On the whole, it appears that an acuity of vision reaching or exceeding $\frac{1}{6}$ has been ultimately attained in 94 per cent. of the operations. These figures have been yielded by patients not only not selected, but in whom some of the precautions ordinarily taken in the flap operation have been laid aside. In some of them, for example, an immature cataract has been removed from the second eye a fortnight after the removal of a mature one from the first. In others the the eyes or eyelids were in various ways diseased, and the operation itself was modified in certain details. As the result of this experience, v. Graefe concludes that, in eyes favourable for flap extraction, the prospect of success is certainly equally great by the new method; and that, in eyes unfavourable for flap extraction, the prospect of success is considerably greater. Considering also that modified linear extraction diminishes the inconvenience to the patient and the duration of treatment, the author is of opinion that it may be accepted and practised to the entire exclusion of the flap method of operation.

After some observations on the position of the patient and operator, and on the direction of the light, v. Graefe proceeds to discuss the use of chloroform. He employs this agent in only a small number of cases (7 per cent.), and less frequently now than formerly. He thinks that a moderate and controllable action on the part of the ocular muscles promotes the escape of the lens, and that the power to make the patient "look down" facilitates the removal of cortical remains. On the other hand, chloroform is of essential service when the action of the ocular muscles is spasmodic or excessive, either on account of mechanical conditions, such as unusual tension of the lids, prominence of the globes, or blepharophimosis, or on account of extreme reflex irritability, or of want of self-control. The surgeon must therefore give or withhold chloroform in accordance with the state of the eye and its appendages, viewed in relation to the general condition of the patient.

The spring speculum in common use has been modified by v. Graefe by bending it backwards towards the temple of the patient, so that it is not in the way of the operating hand. Professor v. Welz has further improved the instrument by adding two little branches to the palpebral portions, by means of which it can be grasped and controlled with facility, and can be removed from the eye with greater delicacy than before. Stress is laid on the position of the fixation forceps, which should seize the conjunctiva close to the corneal margin, and exactly in a line with its vertical meridian. With regard to the sclero-corneal section, the directions formerly given remain in force; and with regard to the formation of a conjunctival flap to the incision, the following conclusions are stated: 1. The presence of a conjunctival flap, covering the whole length of the wound, certainly promotes rapidity of healing and firmness of cicatrix, but plays a very subordinate part in the ultimate result of the operation. 2. Slight differences in the size of the conjuntival flap are wholly unimportant, but it is better that its centre should be sloped a little downwards, and not cut to a point running upwards. The latter form is apt to become rolled, and to occasion swelling and discomfort. 3. Very large conjunctival flaps, extending more than a line and a half from the corneal margin, or reaching towards the reflection, are to be avoided. They occasion bleeding, infiltration of the submucous tissue with blood, trouble from coagula, and difficulties in the removal of the lens and cortical fragments.

With regard to the iridectomy, v. Graefe's experience leads him to insist very strongly upon its being complete up to the angles of the wound. If, from any cause, points of iris tissue are left projecting into the wound, an endeavour must be made to cause them to retract into the anterior chamber, by slight friction upon the eyeball through the closed upper lid, as in prolapsus iridis after flap extraction. In order to accomplish the

complete excision that is desirable, it is well to press a little upon the globe with the closing blades of the scissors. If pointed fragments be left, v. Graefe endeavours to seize and excise them. The drawing out of the iris by the forceps before cutting it must be done carefully and without force; and Mr. Bowman's method of partial tearing from the ciliary margin is recommended with some reservation. v. Graefe appears to doubt whether this or any other method accomplishes the removal of the whole width of the iris; and he cites the case of a patient who died from delirium tremens shortly after linear extraction, and in whose eye he found that a strip of iris, half a millimetre in breadth, had escaped his manipulations. He is careful, before proceeding to the iridectomy, to turn down any conjunctival flap that may have been made, in order to avoid any possibility of curtailing it.

If, after the iridectomy, there be bleeding into the anterior chamber, it is desirable to wait a little before proceeding to open the capsule, since the presence of blood interferes with the accurate performance of this step of the operation. If the closed lids be lightly pressed upon with a pad of charpie for a minute or two, the bleeding will usually cease, and then slight pressure on the sclera above the section will cause the escape of the blood. During the opening of the capsule slight pressure should be made on the globe with the fixation forceps, so as to steady the lens, and to offer the necessary resistance to the cystitome. In order to insure complete laceration, the cystitome may be carried round the equator of the lens, a little over its posterior aspect.

For the removal of the lens, v. Graefe states that it is in every case *possible* to dispense with any form of traction instrument, and to accomplish the desired end by the gliding pressure of the back of the spoon upon the sclera above the wound. The advantages of so proceeding are a less liability to loss of vitreous (in the ratio of 4 to 10), and a more complete escape of the cortex. On the other hand, when the lens is hard, its removal by pressure only takes more time, and therefore protracts the tension of the wound and of the iris, and occasions a larger per centage of cases of delayed healing. The use of the traction-hook is therefore to be recommended when the lens does not readily yield to pressure, and will be required in about one operation out of eight, To determine its necessity must be a matter of tactics and experience; but v. Graefe mentions as a chief indication an arrest of the progress of the lens—a stopping or sticking fast on its way. In such cases he still thinks his hook the best traction instrument that has been devised. When the lens is escaping regularly and uniformly under pressure, but threatens to lose the lower part of its cortex, he places the back of the spoon on the lower part of the cornea, and makes a gliding pressure upwards.

The lens being removed, the patient should turn the eye upwards, a movement that promotes the closure of the wound, and that facilitates the removal of the spring speculum.

Finally, it is necessary thoroughly to clear the pupil of cortical masses, and to press down the conjunctival flap into its proper position. After waiting a minute, renewed pressure should be used, in order to empty the chamber of its small resecretion of aqueous humour, by which some small cortical fragments will be floated away in suspension.

The uninterrupted healing of the great majority of the cases has allowed of an extremely simple after-treatment. The compressive bandage has been applied on the operating table, and the patient led away to his ward. Sometimes, on account of the pain occasioned by the fixation forceps, and by the conjunctival wound, cold moist compressors have been applied for the first ten or fifteen minutes, and then replaced by the bandage. This is at first drawn somewhat tightly, in order to procure perfect adaptation of the wound, and to check any tendency to bleeding. The bandage is allowed to remain undisturbed for about six hours. The second bandage, somewhat less tight than the first, to allow room for transudation, but still tight enough to close the eyelids, and to keep them and the globe at rest, may remain from about the sixth to about the sixteenth hour. The third, again, is drawn more firmly, and especially firmly if there be any appearance of swelling. The period from the sixteenth to the thirtieth hour is the most critical, and firm pressure is then of especial value. During this period, v. Graefe makes a fourth ascending turn of the bandage. It is necessary, after linear extraction, to make the second and third turns of equal tightness, and not, as in flap extraction, downwards, to have the second somewhat loose and the third tighter. This method, which closes the downward flap, would tend to open the upper linear section. When there is a plentiful secretion of tears, the compress must be renewed more frequently than usual in the time immediately following the operation; but such plentiful secretion, commonly associated as it is with periodically increased tenderness to touch, is usually stopped by a night's rest, obtained, when necessary, by a morphia injection.

Although the chief purpose of the bandage is fulfilled in the first thirty-six or forty-eight hours, it should be retained for four or five days and laid aside gradually, first for an hour or so in the day, then for a longer period, then for the whole day, and lastly at night. When an eye has become accustomed to a certain pressure, its sudden and complete removal may produce a congestion, followed by hurtful consequences; and it is well known that the ocular vessels are always more distended during the hours of sleep.

Although after linear extraction there is less necessity than after the flap operation to keep the patient at rest, yet still there must be no want of caution in this particular. Opening and inspection of the eye should be avoided when all is well externally, and only practised by the aid of a wax light and focal illumina-

tion, when swelling or other bad symptoms demand careful scrutiny. Atropine should not be instilled until the third day, and then once or twice daily. If it produce irritation of the conjunctiva, a weak solution of acetate of lead may be used alternately with it.

If the section pursue an abnormal course, the treatment is much the same as after flap operation. If within six hours the pain of the wound has not subsided, a morphia injection may be given; and if there be much lacrymation, cold compresses may be applied for five or ten minutes at each change of the bandage. Continuous cold will endanger the healing process. In plethoric persons, who are accustomed to lose blood, continuing pain may require venesection, followed by the morphia injection. In other cases, when the morphia does not remove pain, and the patient is not very feeble, an active purge may be given—ten or fifteen grains of calomel, with as much rhubarb, followed by a tablespoonful of castor oil, if there be no evacuation within eight hours. These means will only be of service when they anticipate the more marked symptoms of mischief, such as profuse secretion and actual swelling of the lids. If these symptoms once appear, the only available treatment is to apply the compressive bandage firmly, to change it as often as the amount of discharge may require, and to apply warm aromatic poultices at each time of changing. At the same time the general condition of the patient may be met by appropriate treatment, either antiphlogistic or supporting.

It is not one of the least merits of the linear extraction that the eye is much sooner out of danger by this method than by the flap operation. In the former the risk of suppuration scarcely extends beyond the thirty-sixth hour. In the latter it extends even to the sixth day. In v. Graefe's practice, suppurative panophthalmitis occurred for the first time in the hundred and fifth operation.

In the cases of partial inflammation, in which the formation of pus is limited in extent, the same treatment by bandaging and aromatic (camomile) poultices must be employed, with morphia injections if the pain be severe and destructive of rest. When the acute stage is past, the effects may require atropine instillation, or sometimes leeching and mercurial friction.

When a slight and sub-acute or chronic irritation is set up, sometimes due to the entanglement of a bit of iris in the wound, sometimes to imperfect coaptation of its edges, sometimes to cortical *débris*, sometimes to a premature removal of the bandage and relaxation of care, it is necessary to continue the ordinary treatment longer than usual, and sometimes (after the third or fourth day) to use leeches. Any projecting portion of iris should be cut off, and the aqueous humour, if turbid, evacuated by puncture. Once only has v. Graefe found it necessary to make a downward iridectomy for the cure of such irritation.

A number of rare accidents are next mentioned and discussed, and the Professor then goes on to the subject of residual capsular opacities. He counsels great care in operating upon them, a delay of at least four months, unless there be increased tension, and a very clear prospect of improvement to sight as an essential condition for interference. A few pages are then devoted to escape of the vitreous humour, and among other possible results of this occurrence, it is said that the presence of vitreous humour in the wound may be a source of delayed healing and irritation, and therefore of danger to the result.

The death from delirium tremens, already mentioned, afforded evidence of the exact apposition of the margins of the wound in linear extraction. After flap extraction, the margin of the flap always falls a little below the level of the part from which it was severed.

With regard to the best time for the performance of a cataract operation, there are several circumstances to be taken into account. Sometimes failing vision interferes with the power of the patient to earn a living; and it is necessary to operate earlier than one would choose to do under more favourable circumttances. Sometimes failing vision affects the spirits and health so much that on this account an early operation is desirable. But, *ceteris paribus*, and with some exceptions, we say that both unripe and overripe cataracts are unfavourable for flap extraction. v Graefe thinks that unripeness is of far less importance as an objection to linear extraction; although he does not deny that the transparency of portions of the lens may place a difficulty in the way of entire removal. The practice based upon his present experience is as follows:—

1. When with monocular cataract-blindness the second eye is completely healthy, extraction should only be undertaken, in patients above the age of 50, under peculiar circumstances, or at their urgent desire; and never in the grey-headed. With young or middle-aged people it may be undertaken even before the cataractous eye is quite blind.

2. If cataract be so far advanced in the second eye that the patient can no longer read rapidly, the operation may be performed without fear upon the eye first affected, even if its cataract be be not quite mature. If, in spite of commencing cataract in the second eye, the patient can still read, it is then desirable to wait for the maturity (but not over-maturity) of the first.

3 When both eyes are nearly equally affected, it is unnecessary to condemn the patient by delay to the suffering attendant on increasing helplessness. The worst eye may be operated upon when its cataract approaches maturity; and the other eight or fourteen days later, when the result of the first operation is declared.

4. The same principle applies to the very gradual laminar

cataract formations; and to posterior cortical and polar cataracts when they exist in both eyes, and interfere seriously with vision.

5. When one eye has made a good recovery from the operation, the second, in a healthy patient, may be treated boldly, and its cataract removed in an early stage. There will be much better vision with two eyes from which cataracts have been removed, than with one such while the other is still cataractous.

The paper concludes with some account of the influence of age, health, and various conditions of the eye, upon the results of the operation. Upon these points, of course, larger statistics will shortly be forthcoming.

REPORT OF THE IN-PATIENTS TREATED IN THE ST. PETERSBURGH EYE HOSPITAL IN 1863.

By Drs. Blessig, Magauly, Weyert, and Börling. Petersb. Med. Ztschr. 1865.

This report treats of the cases of 544 patients. During the same year 6,684 out-patients were also treated; but the authors reject them from consideration, as not having been sufficiently under observation for statistical purposes. We select some of the most important statements.

There is a description of an interesting case of diphtheritis conjunctivæ occurring in a boy four years old, previously in perfect health, and terminating in death on the tenth day.

"Notwithstanding the use of iced compresses and calomel, the morbid process spread to the connecting tissue of the eyeball as well as to the skin of both eyelids. An ulceration appeared upon the under lip, and the wound from a previously applied blister behind the ear also shared in the disease. The child sank very rapidly, although all means of support were used."

207 persons (101 males and 106 females) were treated for trachoma and its consequences. 17 were between 5 and 14 years; 58 between 15 and 24; 99 between 25 and 39; 28 between 40 and 59; and 5 between 60 and 79. In only 14 cases the cornea and eyelids were free from consecutive disorders. Trichiasis or entropium was present in 193, almost always with pannus, opacities, and so forth. The treatment was mostly surgical; and in 100 cases operations upon the lids were performed. Iridectomy was required in 2 cases, and paracentesis in 16, on account of corneal ulceration.

In 39 cases of corneal ulcer and hypopyon (27 males, 12 females), von Graefe's treatment by atropine, compressive bandage, and warm fomentations, was almost invariably successful. Paracentesis was required 28 times.

Necrosis of the cornea occurred three times in both eyes.

Two of the patients were children, all were marastic and strumous. The disease was painless, but uninterruptedly progressive.

Several cases of iritis, choroiditis, and retinitis are carefully described; all of them interesting, either on account of the ophthalmoscopic appearances or of the treatment. They do not admit of being condensed.

Iridectomy and iridenkleisis were each performed twice for lamellar cataract (*Schichtstaar*) with good results. In another case, the patient being unruly, the capsule was wounded; and the resulting traumatic cataract was afterwards removed by discission. Six operations were performed for soft cataract; in two cases with unfavourable results. In 24 operations for hard cataract it was found that an iridectomy did not increase the average of success of the flap extraction.

Among the *injuries* a curious case is related, in which a splinter of iron had penetrated the cornea, and was pushed quite into the anterior chamber in the first attempt at removal. The chamber was twice opened, and iridectomy was performed, but the piece of iron could not be found. After a month it became incapsuled by effused lymph; and vision was perfect.

CHROMIDROSIS OF THE EYELIDS.

Professor Rothmund (Monats, Bl. f. Augenheilk, March to May, 1866) records two examples of this so-called disorder. They were both due to the adhesion of atmospheric dirt to eyelids rendered greasy by an excess of sebaceous secretion. The patients were young girls, living and working in a smoky atmosphere. The author believes that other cases recently reported have been really of the same character.

ON THE ORIGIN, SPREAD, AND PROPHYLACTIC TREATMENT OF TRACHOMA.

By Dr. H. Fischer (*Allg. Mil. Arztl. Zeitg.*, 1865) *and Dr. V. Hoffmann* (*A. A. O.*).

Dr. Fischer attributes the so-called military ophthalmia entirely to the overcrowding of barracks, and to the generally defective arrangements therein, and he cites many facts in support of his opinion. When there is no purulent discharge, he strongly recommends that the men attacked should be dispersed in the neighbouring country; but the presence of pus renders it necessary to enforce strict confinement to hospital. For the prevention of the disease he relies upon proper sanitary arrangements, and upon strict cleanliness, both of person and dwelling. For the prevention of its extension when existing, he advises isolation

of the sufferers, careful regular inspection of the healthy, the setting up of hospital tents, careful treatment of all occurring cases, and strict watchfulness over convalescents.

Dr. Hoffman reports an interesting illustration of the effect of removal to a better ventilated place in checking an outbreak of military ophthalmia. The Austrian garrison of Mainz, about 5,000 strong, had 100 men invalided from various forms of eye disease, at the beginning of 1864. By March this number increased to 196, without any marked prominence of trachoma, and with an isolated case of purulent ophthalmia. In April and May there were 12 purulent cases; in June, 23; in July, 47. There was then a decrease, but the disease again gained ground in October, when there were 29 purulent cases, and two or three invalids from eye disease in every company. In November the troops were moved into airy and spacious quarters. In December there were 20 purulent opthalmiæ among 112 catarrhal. In January, 1865, only 5 purulent cases; in February, 2; and in March only 1. The number of other ophthalmiæ, catarrhal and granular, was at the same time reduced to 10 cases, and the epidemic was at an end.

BRAZILIAN OPHTHALMIA.

Dr. Gama Labo, of Rio, communicates to the "Monats Bl. für Augenheilk:" (March—May, 1866), through Dr. B. Ullersberger, his observations on a peculiar form of ophthalmia seen in the Brazils in debilitated persons, reduced by previous disease, and most commonly in slaves and slave children. The disease has three stages. In the first, the conjunctiva of the globe and lids is bright red, the lids are not swollen, the corneal epithelium is disturbed, and the optic disc is reddened, and, as if covered by a transparent veil. In the second stage the cornea appears normal, the reddened palpebral conjunctiva is studded with little elevations, the ocular conjunctiva is whitish gray, with slight wavy wrinkling of its surface, and with the dull red vessels scarcely visible, except in the reflexions. The nerve disc is no more reddened and sharply defined; and its vessels appear whitish red. The moisture of the eyes has a kind of fatty aspect. In the last stage the conjunctiva is dry, and more strongly wrinkled. In the centre of the cornea is a roundish ulcer, surrounded by a belt of purulent infiltration. The anterior chamber contains pus. The patient keeps the eyes constantly closed. Dr. Gama Labo relates four cases in detail, all of which terminated in death.

ON SUB-CONJUNCTIVAL INJECTION OF A SOLUTION OF CHLORIDE OF SODIUM, TO PROMOTE THE ABSORPTION OF CORNEAL OPACITIES.

Prof. Rothmund ("Monats Bl. f. Augenheilk." March—May, 1866), has been employing a sub-conjunctival injection of a solution of chloride of sodium (a scruple to an ounce of water) to promote the absorption of the diffuse corneal opacities left behind by parenchymatous inflammation. In six cases he believes that he has obtained more speedy results than any other known means would have yielded. The solution is warmed, and is very slowly injected by a syringe with a curved nozzle, through a puncture about a line and a half or two lines from the margin of the cornea. The immediate effect is to surround the cornea with an elevated ring like that of chemosis. Under a compressive bandage, the swelling disappears in five or six hours, and the resulting irritation in five or six days, after which time the cornea begins to clear from the margin. After three or four weeks the injection may be repeated; and after from three to five injections, the formation of an artificial pupil has been practicable.

CASE OF HEREDITARY IRIDEREMIA, FROM THE CLINIQUE OF PROF. REUTE.

Reported by Dr. Paul Schröter (*Monats. Bl. f. Augenheilk, Mar.—May*, 1866).

In a woman, 42 years old, from Wildenfels, who attributed the state of her eyes to her mother having been frightened by an amaurotic man when pregnant with her. The following condition was observed :—

Both eyeballs small, deeply sunken, and the subjects of alternating squint and of nystagmus. *Right eye:* Cornea small, with diffuse turbidity at its upper margin; iris wanting; lens with lamellar turbidity, small central opacity, and lamellar streaks. With a 12-inch concave, No. 17 was read at 12 inches. *Left eye:* Cornea small; iris wanting; anterior capsule white and densely opaque, except at one outer portion, through which turbid lens substance could be seen. Fingers could only be counted when very near. The opacity of the lens had shown itself only a few weeks previously. In early childhood the sight was very imperfect, but it improved whilst at school, so that small print could be read, and there was at the same time good distant vision.

This woman had two children, and the eldest, a girl seven years and a half old, was born with the same deficiency. In this child the whole corneal circle appeared perfectly black. The experiment of Purkinje gave only the first (erect corneal)

image; but the margin of the lens could be distinguished with the ophthalmoscope. The absence of the image from the anterior surface of the lens was due to this surface, in irideremia, being in contact with the cornea. The image from the posterior surface was wanting in both eyes, on account of some turbidity at the posterior poles. In the anterior chamber of the left eye there floated a white thread-like body; the characters of which could not be ascertained, on account of the great nystagmus. The child was not dazzled, and saw large objects well; but small ones with obvious difficulty, and only in a good light. Both mother and child were subject to attacks of headache.

CASES OF ISCHÆMIA OF THE RETINA.

By Prof. Rothmund (*Monats. Bl. f. Augenheilk, Mar.—May*, 1866.)

A healthy country girl, 18 years old, in service, had observed, six days before, that all objects became suddenly obscured, and, on the next day, she became perfectly blind. Externally there was nothing noticeable beyond extreme dilatation of the pupils. The central artery of the retina was very small at its point of emergence, and its peripheral branches were completely empty. The veins were dark and also somewhat narrowed. No pulsation could be produced by pressure. The face was flushed, pulse full, heart sounds normal; there were noises in the ears, and feeling of heat in the head. A derivative treatment was employed (leeches, sinapisms, footbaths, and calomel). After three days the patient could see the movements of a hand with the left eye; the right remaining blind. After nineteen days, in which no improvement took place, paracentesis corneæ was performed on the right eye. On the following day she could count fingers with the right eye at six inches; and, after three days, at eighteen inches. The action of the pupil was also restored. Paracentesis was then performed on the left eye; with which, nine days later, the patient read No. 17. From this time the sight steadily improved, on the left side more rapidly than on the right; and at the end of eight weeks the retinal arteries were naturally filled, and vision on both sides was perfect.

The second case was in a girl of 13. The history is only that she is very anæmic, but otherwise healthy, and that she became suddenly blind four days before being brought for treatment. With the right eye fingers could be counted at four inches; with the left there was only quantitative perception of light. The retinal arteries on both sides were empty. Parancentesis was performed on both eyes; and, on the following day, fingers could be counted with both at four feet. In eight days there was no further improvement, and the paracentesis was repeated.

From this time the sight mended daily, and was quite restored in eight weeks.

The author observes that in this affection the operative treatment, first advised by Alfred Graefe, and decidedly the only trustworthy method, is only serviceable when the anæmia of the retina depends on a mechanical cause. In two other cases he performed paracentesis and iridectomy without result, although the retinal vessels were quite empty. But, in one of these cases, subsequent events showed the presence of a tumour in the brain, and in the second, the anæmia of the central artery was only the first step in degeneration of the retina dependent upon Bright's disease.

ON A PLANO-CONVEX LENS, WITH MEASURING SCALE FOR OPHTHALMOSCOPIC INVESTIGATION.

By Dr. Colsmann.

Dr. Colsmann has published (Deutsch. Klin. 1866) a report of his ophthalmic hospital at Langenberg, near Elberfeld. It contains nothing of special interest, except an account of his use, for ophthalmoscopic investigation, of an object glass of two-inch focal length, plano-convex, and with a scale of millimetres on its plane surface. Holding this lens in the ordinary way, two inches from the eye of the patient, he finds it easy to make any part of the inverted image coincide with the scale, and the measurements thus afforded supply a ready test of the state of the refraction. When the eye of the observer is distant from six to nine inches from that of the patient, the image of the optic disc of an emmetropic eye has an apparent diameter of from five to seven millimetres. The image of the disc of a myopic eye has an apparent diameter of from two to three millimetres, and that of a hypermetropic eye from nine to ten. This great apparent enlargement of the disc is especially manifest in elderly hypermetropic people, in whom the error of refraction is not concealed by accommodation.

CASES OF HEMIOPIC CONTRACTION OF THE FIELD OF VISION.

By Dr. Gunning.

Dr. Gunning, in a report of his ophthalmic hospital at Amsterdam (Nederl. Tijdschr. v. Geneesk. II., 1865), describes the following cases:—

In a woman 52 years of age, who had had an attack of apoplexy nine weeks previously, and had recovered from the resulting paralysis of the left side, vision was totally lost in the inner half of the field of the right eye, and in the outer half of

the left. The vertical line which sharply defined the defect, was situated in the right eye inwards, in the left outwards, of the yellow spot. Central vision was retained, but the patient could only find her way about with difficulty.

The second case was in a man 50 years old, who was attacked with intense headache in the street eleven years previously, and who then, although he did not lose consciousness, could not find his way home. When seen by Dr. Gunning he had no ailment beyond some dullness of perception, and loss of the right half of the field of vision in both eyes. In the lower quarter of the defective regions there was still some perception of light.

In neither case could anything abnormal be discovered by the ophthalmoscope.

MR. MARSON ON VARIOLOUS OPHTHALMIA.

Mr. Marson, our chief English authority in all which relates to small-pox, gives us some very interesting information on variolous ophthalmia in his admirable article, just published in Reynolds's System of Medicine. He states that in 1838, having then seen upwards of 1,500 cases of small-pox, he had never once witnessed the formation of a pustule on the eye. Since that date, however, he has several times seen them. Out of 15,000 cases treated at the Small-pox Hospital during thirty years, the formation of a primary pustule in the eye was observed only in thirty-six instances, and in not one of these was the eye in any way injured. The explanation of the immunity from bad results is, that the pustule occurs on the thickest part of the conjunctiva, and never on the cornea. Its usual position is midway between the cornea and the inner canthus, sometimes near the outer canthus.

The ulcers of the cornea occurring in connection with small-pox, never take place during the development of the eruption. They occur during the stage of secondary fever, sometimes as early as the tenth day, sometimes as late as the thirtieth, most frequently at about the fourteenth. These dates are calculated from the commencement of the eruption. The ulcer usually forms at the margin of the cornea, and spreads with greater or less rapidity in proportion to the severity of the secondary fever.

Sometimes ulceration commences simultaneously at opposite parts of the cornea, and when this is the case it spreads very rapidly, and the eye is almost inevitably lost. Occasionally the entire cornea may be swept away within forty-eight hours of the apparent commencement of the ulceration, and now and then this goes on without causing any inconvenience to the patient.

"This destructive ulceration never goes on rapidly but when there is a high degree of secondary fever present. That is a point which should be particularly remarked. It is likely to occur when there is a hot and dry state of skin, rapid pulse, thirst, loaded tongue; these having been preceded by a very confluent state of the disease, and the patient has just escaped dying at the usual time, namely, the ninth, tenth, or eleventh day of the eruption. Then it is that some serious consequence may be apprehended, such as the loss of an eye, formation of large and deep abscesses, sloughing of the cellular membrane, or may be formation of matter in one side of the chest. Some of these serious results may be expected when the secondary fever runs high in confluent small-pox, combined with the circumstances above detailed."

Mr. Marson also mentions that conjunctivitis, rather slow in its progress, begins in the course of a tedious convalescence as late as the third or fourth week, and with it there may usually be found a small central ulcer on the cornea. He makes no mention of iritis.

THE DYNAMIC ANOMALIES OF THE LATERAL MUSCLES OF THE EYE AND OF THE ACCOMMODATION.

Lecture delivered by Prof. v. Graefe, at the Clinique of Dr. Giraud-Teulon, and reported by M. Delfau (*Gaz. des Hôp. 29th Nov.* 1866, p. 550. *Annales d'Oculistique*, vol. 56, p. 318).

One of the most important points in the practical study of the impairments of associated vision, and one of the most embarrassing to the surgeon, is the recognition and determination, in every case, of the mutual relations which connect the convergence of the optic axes, and the accommodation of each eye. It is, therefore, of some interest to examine the practical means of investigating these relations.

Direct binocular fixation and exact accommodation are subject to some anomalies, and in some patients, for example, these functions are accomplished with an excess of force.

In dynamic divergent strasbismus direct binocular fixation is possible for every distance; but only by an exertion of muscular power.

1. Take a patient in whom the near point is at two inches, and the far point at infinity. If prisms, with their bases outwards, are placed before his eyes, he will still have distinct vision, but only by a muscular effort on the part of the internal recti. He is in the same position with a patient in whom the lateral muscular equilibrium is destroyed; that is to say, who suffers from *dynamic divergent strabismus.*

2. Take another person, with a perfectly normal eye, and place before it a concave lens. He will still be able to see from

infinity to about four inches, but only by an effort of accommodation. Certain patients fulfil pathologically the conditions of this experiment, and the two foregoing types were taken by the Professor as the subject of his lecture.

I.—*Failure of Equilibrium of the Lateral Muscles of the Eye.*

Diagnosis.—There are two means of detecting loss of equilibrium of the lateral muscles. The first is to bring a small object, as the point of a pencil, to within two inches and a half or three inches of the eyes, and to direct the patient's attention to it. It will be observed that the convergence of the eyes is accomplished with an effort, and that the patient has a tendency to draw back in order to require a less degree of convergence. Then, if we place a hand so as to conceal the object from one eye, that eye will deviate outwards, placing itself in a state of divergent strabismus.

The second means is by placing before one eye a prism with its base superior, when the difficulty of convergence will be increased.

Prisms, however, are of much utility when the insufficiency is not very great in degree. They may be worn of three or four degrees on each side; but the actual remedy is tenotomy.

II.—*Excess of Accommodative Power.*

At present we recognise faults of accommodation by two methods:—

1. By the state of the refraction;

2. By the absolute latitude of accommodation. To these Professor v. Graefe would add:—

3. The relative latitude of accommodation. The first two means furnish certain data; but they do not discover whether the patient makes an effort of accommodation at the distance at which he works. Professor v. Graefe proposes the following test. If a patient is exerting an effort of accommodation at eight inches, and we cover one of his eyes, the eye that is covered will make a movement of convergence. Prisms with their angles superior or inferior will display this convergence; just as, in loss of the equilibrium of the lateral muscles, they display the dynamic divergence. In the former case (convergence) the double images would be crossed; in the latter, homonymous.

This method is sometimes imperfect, and should be combined with a measurement of the amount of accommodation that the patient is exerting.

But, on placing a convex glass before the eye, a less amount of accommodative force is called forth; an amount correspond-

ing to the strength of the glass being put in reserve. On employing, on the contrary, a concave glass, an effort of accommodation proper to counterbalance its dispersion is called forth.

This being so, having ascertained the strongest convex and the strongest concave lens through which a patient can see distinctly at a given distance, we have the accommodative force that is put in reserve, and that is brought into play. This is the relative latitude of accommodation.

For clinical examination Franklin's glasses are convenient. They consist of two half lenses in juxta-position along a horizontal line. They may be + 20 and — 20; or even 7, 6, or 5.

In near vision one eye is excluded, in distant vision both are used, but by straining the internal recti.

It is precisely because the patient is obliged to make an effort with the internal recti that he would require a stronger concave glass.

Finally, as regards the refraction, we observe, in such cases, an exaggeration called dynamic refraction; emmetropia may simulate myopia.

4. The worst consequence is the increase of the myopia. Under such conditions we see, in fact, that the myopia has a progressive course. Its progress has been attributed chiefly to the movements of convergence, which produce compression of the eyeball and choroidal congestion. Professor v. Graefe does not deny this cause; but he does not think it the chief one. It is not the normal convergence which chiefly augments the myopia, but the abnormal, or exaggerated. It is this which constitutes the gravity of myopia.

In 32 myopes, Professor v. Graefe has cured the defect of equilibrium, and in only two of these cases has the myopia afterwards increased. In the other 30 it remained stationary.

Treatment.—The defect of equilibrium may be treated by three methods:—

1. By acting upon the muscles (tenotomy).
2. By acting upon the accommodation by concave glasses.
3. By acting upon the convergence by prismatic glasses with the bases inwards.

Of the two last means, the only effectual one is usually the employment of prisms, which remove to a greater distance the virtual crossing of the optic axes, relieve the adduction, and thus re-establish the harmony between the powers of accommodation and convergence.

With regard to concave lenses, they are inconvenient by diminishing the retinal images, 1st, by the greater distance to which the object is removed; 2nd, by their own optical properties. This defect often destroys the benefit of the use of concave lenses for short distances; the patient, notwithstanding all

advice, having an invincible tendency to bring objects close, in order to obtain larger retinal images.

With respect to this, the concave glasses may become a motive for placing a prism with its base inferior before one eye, so as to produce diplopia. If the eye be normal, the two images will be one above the other on the same vertical plane. If, on the contrary, the eye suffers from a defect of muscular equilibrium, the images, still one above the other, will also be separated laterally. The degree of the lateral separation is the measure of the defect of equilibrium.

We must here remember that there is a relation between convergence and accommodation. A certain convergence corresponds reciprocally to a certain accommodation. In acting upon the accommodation by glasses, we act also upon the convergence. A concave glass requires that the accommodation should become greater in order to counteract the greater dispersion of the rays of light.

It follows that, in many patients, the presence of a concave lens before the prism nullifies the action of the latter, and restores the laterally displaced images to their vertical superposition. This is easily understood. The excess of accommodation required by the presence of the concave glass acts secondarily upon the convergence.

Consequences.—The defect of equilibrium of the lateral muscles entails many consequences.

1. *Muscular asthenopia.* The internal recti being fatigued by excess of work.

2. *Neuralgic sensations.* Such as orbital pains, vertigo, nausea, &c.

3. *Various apparent disorders of refraction.* Together with the defect we may doubtless sometimes find emmetropia, but one of the most constant phenomena is apparent myopia. It is discovered on seeking the far point for binocular vision. There is then an apparent myopia, because the patient is unable to relax his accommodation beyond a certain point nearer than infinity, and that corresponds to the condition of his static refraction.

Moreover, we often find an apparent exaggeration of a myopia actually existing. For example, a patient whose far point, for No. 1 of Jaeger, is at eight inches, will be able to read with a lens of $-\frac{1}{8}$, or weaker. When he has defect of equilibrium of the lateral muscles, he requires + 40 and — fixed in the same mounting. The surgeon need not change the glasses to determine the state of dynamic refraction of his patient, but judges from his account of the distance at which they enable him to read.

In some emmetropes there is always an effort of accommodation, even for distant vision. Some persons, in fact, employ three-fourths of their power of accommodation, while others scarcely employ one-tenth.

Professor v. Graefe considers that the energy of accommodation is represented by the accommodative force that the person is able to exert. Thus we may meet with hypermetropic eyes in which the error of refraction is concealed by great accommodative power, and which never suffer from asthenopia.

Treatment. The treatment may be directed to the accommodation, to the muscles, or to the refraction.

1. The accommodation may be reached in an indirect manner, by using prisms with their angles inwards, which leave at the disposal of the patient a greater power of accommodation for any given distance.

2. The muscles may be treated by tenotomy. Professor v. Graefe has practised this.

3. The refraction may be modified by convex glasses. These diminish the accommodative effort, so that they may enable a patient, in fact, to use only half of his relative accommodation instead of the whole.

The choice of glasses calls for certain precautions:—

If the accommodation is limited, the use of prisms with their angles internal would be injurious. It is inconvenient to bring the far point too near; and, by doing so, we cause fatigue and headache.

On the other hand, some persons are in the habit of always exerting their accommodation, and cannot read, for example, if it be relaxed. In such cases glasses which require relaxation of the accommodation (convex glasses) cannot be borne immediately. In this respect, therefore, certain precautions must be observed.

To these inconveniences must be added also the question of excentric vision through spectacles; a neglected subject to which Professor v. Graefe intends speedily to call attention.

Conclusions. From the foregoing we may conclude that defect of equilibrium of the lateral muscles of the eye, as well as defect of equilibrium of the accommodation, impedes the functions of the eye, and may even injure its integrity.

The analysis of these matters should, therefore, be an object of care to the surgeon; and an exact knowledge of them is even more necessary than the determination of the state of the refraction, which, taken alone, leaves the history of the case obscure and incomplete.

THE TREATMENT OF CONICAL CORNEA.

v. Graefe (*Arch. f. O.* xii, 2, pp. 215—222),

After a glance at the methods hitherto employed for treating this condition, mentions that it occurred to him to excite ulceration and cicatrization in the centre of the diseased cornea. It is

known that such a process will produce flattening of a cornea previously of normal curvature; and v. Graefe believed that the same action would be even more pronounced in an abnormal one. Patients with central leucoma in a normally curved cornea usually have much better vision, after a peripheral iridectomy, than is found in conical cornea of a high degree, in which vision often sinks to $\frac{1}{30}$, $\frac{1}{40}$, or even less.

The plan thus suggested was put in practice in the case of Frau. O., 43 years of age. She stated that the sight of her right eye had been very imperfect since childhood; but that the left, although somewhat short-sighted, had had good vision until four years ago. Since then it had gradually and steadily failed, without inflammation, but with some irritability from light. On the 30th of May examination discovered a high grade of conical cornea in both eyes, in the right more than the left. Especially the central regions of the corneæ had assumed the form of pointed cones, of which the apices presented greyish opacities as large as pins' heads. The ophthalmoscope showed moderate ectasia posterior, and excessive irregular astigmatism. Vision is so bad that the patient finds her way about with difficulty. With the right eye she deciphers 16 Jaeger at an inch and a half, but cannot decipher 14; and a stenopaic slit, tried in all directions, is scarcely of any assistance. Excentric vision is also very bad, the patient not counting fingers with certainty in a moderate light. Neither spherical nor cylindrical glasses are useful. In the left eye the sight is somewhat better, the patient reads No. 14, and can be made to read No. 8 by stenopaic spectacles. Selecting the right eye, as the worst, for treatment, in the early part of June, v. Graefe cut away a portion of the central turbid part of the cornea. He made a little flap with a Beer's knife without opening the anterior chamber, and then snipped off this flap at its base with scissors. Two days later the surface of the wound was touched with diluted nitrate of silver, and this cauterization was repeated at intervals of two or three days. The resulting ulcer was perforated in the fourth week with a stilet, and during the next week the closed fistula was reopened every day or every second day. After this it was allowed to heal. The curvature of the cornea was already considerably improved, and also the vision with regard to a general survey of the room. On the 5th of August the patient was discharged, in the following condition. In the centre of the cornea a dense, white opacity, half a line in diameter, surrounded by a slight grey halo, which made the diameter of the whole turbid part slightly exceed a line. At the most cursory glance, but especially by examining its reflections, the improvement in the corneal outline was very visible. The central opaque portion had fallen back almost to its natural position, and the whole membrane departed but little from its proper curvature. The patient read No. 7 fluently at an

inch and a half. The distant vision was $\frac{1}{30}$, but was raised to $\frac{1}{12}$ by a concave cylindrical glass. The clearness of the ophthalmoscopic image was so much increased as to render it probable that vision would have been still better but for the co-existence of a certain degree of amblyopia; and v. Graefe observed that during the last two weeks of the patient's stay, although the cornea continued to clear, there was no corresponding improvement in vision. The left eye will hereafter be treated in the same manner, and the final results carefully noted.

The suggestion to make artificial ulcers for the cure of a morbid ectasia is not new; but it is new, as far as v. Graefe can ascertain, in cases of conical cornea.

EXPERIMENTAL CONTRIBUTION TO THE STUDY OF GLAUCOMA.

By Dr. Wegner (*Arch. f. O.*, vol. xii, 2, pp. 1—22).

In the course of 1866, four cases of neuralgia of the trigeminus, complicated with simple glaucoma, came under the observation of Professor Horner of Zurich, who was led to believe that there must be some relation of cause and effect between the two disorders. He requested Dr. Wegner, then at Zurich, to institute certain experiments. They were carried on in the laboratory of Professor Dubois Reymond, and with the assistance of Dr. Rosenthal. Dr. Wegner begins his paper by a detailed account of two of the cases, and refers in a note to two similar cases already recorded by Mr. Hutchinson, in vols. iv and v of the Ophthalmic Hospital Reports. He then proceeds to say that, in endeavouring to ascertain the influence of certain nerves upon the vessels, and upon a transudation from them increasing intra-ocular pressure, the first step was to determine by what nerves the vessels were supplied. In many young white rabbits the blood-vessels of the iris are perfectly open to observation, and display a considerable breadth, although in older rabbits they are so much covered by the epithelium of the iris as to appear only as lines. With regard to the vaso-motor supply of the iris, authorities differ. Claude Bernard, and those engaged like him, in the study of the nerves of vessels, seem, with the exception of Schiff, to have made no direct observations on the point. Some have assumed that the cervical sympathetic supplies the vessels of the eye, as of the other parts of the head; while Schiff, Funke, and others, declare the trifacial to be the only nerve of the internal ocular circulation, and that the vessels of the iris are independent of the sympathetic. To decide the doubt, the following experiments were tried:—

I. The cervical sympathetic of a young white rabbit was divided on one side. The vessels of the iris on that side immediately enlarged, the iris became hyperæmic, with small extrava-

sations here and there. The upper end of the divided nerve being irritated, the vessels contracted until they were empty; the iris becoming white and bloodless. The irritation ceasing, the hyperæmia returned.

II. The trigeminus of one side being divided, the vessels of the iris became dilated, as in the former experiment. Irritation of the sympathetic produced no contraction.

III. The trigeminus being divided on one side, and the cervical sympathetic on the other, the hyperæmia of the iris was alike on both sides. In many instances, however, the congestion was at first greater, on the side of the trigeminus section, than on that of the sympathetic section. This could be explained on the ground that the division of the trigeminus involved some hyperæmia of the brain, by which the ocular circulation was influenced. Very soon, however, this excess of congestion subsided, and the two eyes remained in the same condition.

From these experiments Dr. Wegner concludes that the vaso-motor nerves of the iris are wholly derived from the sympathetic, but that they pass into the trunk of the fifth, and are carried by it to the eye, as well as divided when it is cut through. In the cranium they seem to lie on the inner side of the fifth, for in one case the section of the latter was incomplete. The vaso-motor nerves were divided, and the cornea and conjunctiva deprived of sensation, but the other parts supplied by the fifth were unaffected. It was afterwards found that only quite the inner portion of the nerve had been cut through. The ophthalmoscope appeared to show that the vessels of the choroid and retina were affected in like manner with those of the iris.

The innervation of the vessels of the iris having been determined certainly, and that of the vessels of the choroid and retina with probability, the next step was to devise a means of accurately determining and measuring the variations of intraocular tension. For this purpose a small conical canula was pushed into the anterior chamber near the outer margin of the cornea. The smaller end of this canula was a solid point, above which was a lateral opening, and the larger end was fitted with a fine stop-cock; above this was fastened to the canula a tube composed of alternate portions of india-rubber and glass, filled with a solution of common salt. This tube made a communication between the canula, and through it the anterior chamber, and a manometer fitted with a capillary mercurial scale. It is unnecessary to describe Dr. Wegner's account of the various precautions required to avoid erroneous conclusions. His experiments seem to have been conducted with due caution. He found that in different rabbits the degree of intraocular pressure varied within rather wide limits, as from 18 to 35 millimetres of mercury; and he found also that a large iridectomy in one

eye, after a lapse of sixteen days, and complete healing of the wound, invariably reduced the pressure of the eye operated upon, as compared with the other eye of the same animal, by 6 or 7 millimetres.

The working of the instrument having been thus tested and mastered, the cervical sympathetic was laid bare, and the canula introduced into the eye. As soon as the mercurial scale was at rest, the sympathetic was divided. This was done repeatedly, and the pressure was always diminished from 4 to 8 millimetres, the mercury sinking gradually. At the same time trials were made to discover the effect of atropine on intraocular pressure. In many experiments it was always found that atropine, whether applied locally or given internally in corresponding quantities, considerably diminished the tension of the globe. It might hence easily be inferred, if the dilatation of the vessels by section of the sympathetic diminished tension, and the effect of atropine was also to diminish tension, that atropine must exert a generally paralysing influence over the vaso-motor nerves. The diametrically opposite statements of Fleming, Jones, and Hayden, did not deter Dr. Wegner from pushing this inquiry. He injected half a cubic centimetre of a solution of one part of atropine in one hundred parts of water under the skin of the ear of a rabbit. In a short time the vessels of the ear became distended, its temperature elevated between two and three degrees (Cent.) above that of the other ear, and irritation of the cervical sympathetic produced little or no effect in contracting the vessels; so that the peripheral extensions of the nerve-fibres, or the muscles of the vessels themselves, were paralysed by the atropine. Persons examining the rabbit, and not knowing what had been done, believed that it had been subjected to Bernard's experiment. The same paralysis of the muscular wall of the vessels was observed in the iris after the instillation of atropine.

From this Dr. Wegner concludes that the diminution of intraocular pressure after the section of the sympathetic, and after the use of atropine, is in both cases due to the paralysis and dilatation of the vessels, and that the clinical observation of v. Graefe, "that atropine exerts a tranquillising influence over the nerves of secretion of the eye" is substantiated by exact experiment. He explains the diminution of pressure thus:—The pressure to which the blood is subjected in any artery is the result of two factors—the stroke of the heart and the resistance of the arterial wall. The latter may be divided into two portions, the passive, the elasticity of the arterial wall, and the active, the contraction of the arterial muscles. The latter forms no inconsiderable part of the whole, and, if arrested, the pressure of the blood is diminished. This diminution is shared by the general pressure, in a manner more or less marked according to the degree of extension of the muscular paralysis. Shortly after the

paralysis of the arterial muscles, the blood would flow in unusual quantity along the dilated channels, and the capillaries would be distended, at first under increased pressure. This would soon cease, the capillaries being neither elastic nor contractile, and opposing no resistance. The blood would soon, in the whole region of the paralysed artery, flow through dilated channels under diminished pressure.

An experiment of another kind gave additional evidence of the change produced in the ocular tension by section of the sympathetic. After the nerve was divided on one side in a rabbit, a traumatic iritis was excited in both eyes at the same time by similar injuries. It was evident to the most unpractised observers that the eye on the side of the section was far less hard than the other, and the difference was also shown by the manometer.

It was evident that section of the trigeminus would have necessarily the same influence, with regard to tension of the eyes, as section of the sympathetic, since the vaso-motor nerves of the choroid and iris would be divided in either case. Moreover, Cl. Bernard and Donders have already remarked that, after section of the trigeminus, and prior to the occurrence of inflammation, the eye on the side of the section is considerably softer than the other.

Since the ocular tension is diminished by paralysis of the vaso-motor nerves, it would probably be increased by irritating them. Dr. Wegner found it extremely difficult to bring this to an experimental test. In four trials, the results were twice negative, and twice an increase, for a short time only, of from 1 to 2 millimetres. The case is very different where there is pathological irritation without operative interference, especially when a somewhat slight irritation is long continued. Dr. Wegner thinks himself authorized to conclude that the normal intraocular pressure is regulated by the sympathetic filaments that supply the vessels of the iris and choroid, and that, as the essence of glaucoma and the starting point of all its phenomena is to be found in increased tension, so the cause of glaucoma must be sought in a pathological irritation of the sympathetic fibres of the eye, either idiopathic or reflex in its character.

Returning to the recited cases of neuralgia complicated with glaucoma, Dr. Wegner expresses his belief in a causal connection between the two diseases, and suggests for this three possibilities. Either the filaments of the sympathetic shared in an inflammatory process, or they were irritated by pressure, or by reflex action through some fibres of the trigeminus proceeding from the interior of the eye. The possibility of the last event was shown by the following experiment:—

The posterior auricular nerve of a rabbit was exposed and divided, and the hyperæmia and elevation of temperature, de-

scribed by Schiff, were produced. The central end of the nerve was irritated, and both ears were observed. With evidences of acute pain, the arteries of both ears became contracted to complete disappearance of their calibre. The contraction was maintained while the irritation continued, and was soon followed by complete dilatation when the irritation ceased. All reaction of the vaso-motor nerves to irritation was stopped by section of the cervical sympathetic. The same results were obtained in the ear when the irritation was applied to the temporo-auricular branch of the trigeminus, or to the peripheral terminations of this nerve in the lip or the conjunctiva; and in the eye, by irritation of the central end of the supra-orbital nerve.

It would therefore appear that the vaso-motor, like the sensitive and the musculo-motor nerves, may be excited by reflex action from various points of the surface. Their centre of reflex action is probably in the spinal cord. Yet in many instances the arrangements seem to be such that only a local reflex action of the vaso-motor nerves follows from sensible irritation. In one case observed by Dr. Wegner, irritation of the posterior auricular nerve only produced contraction of the arteries of the ear on the same side, on whichever side it was practised. It must be conceded, therefore, that the glaucoma under consideration may have been produced by reflex action through the fifth nerve.

The fact of a reflex excitation of the vaso-motor through the sensitive nerves seems not to be unimportant with reference to many well-known phenomena; such as the pallor or redness produced by mental emotion. It will also help to explain many things both in general and in ophthalmic pathology, such as amaurosis occurring during neuralgic paroxysms, the recurrence of iritis from pupillary adhesions, and the occurrence of sympathetic ophthalmia.

BROWN CITRINE OINTMENT IN THE TREATMENT OF TARSAL OPHTHALMIA AND PHLYCTENULAR CONJUNCTIVITIS.

Dr. E. Williams (Cincinnati), in the Transactions of the American Medical Association, speaks in the highest terms of a *brown citrine ointment* in the treatment of these troublesome affections. He thinks it more efficacious and less irritating than Pagenstecher's ointment of precipitated yellow oxide of mercury. All crusts being first softened and removed from the eyelashes, and the eyelashes cut close if necessary, the ointment should be gently applied to the tarsal margins with the pulp of the finger, in most cases every night. If it occasion swelling, it should only be applied every other night. In cases of phlyctenulæ it may be applied, as other ointments, with the point of a camel's hair brush. Its use should be continued, once or twice a week, for

some little time after recovery, in order to prevent relapse; and it may also be applied, with great benefit, to the eruptions of the face and head with which the ocular affections in question are often associated. When there is much general conjunctivitis, Dr. Williams uses also a lotion, containing four grains of sulphate of morphia, and half a grain of sulphate of copper or of zinc, to the ounce of distilled water, to be used three times a day. If there be much photophobia, he precedes the application of the ointment by the instillation of a solution of atropine, using one grain to the ounce of water for children from one to five years old, and two grains to the ounce for children above five years. These solutions are applied from three to six times in the twenty-four hours; and quinine is also given in full doses, as one or two grains three times a day. If this method does not speedily diminish the photophobia, he combines opium with the quinine, giving from half a grain to a grain for a dose. As soon as the acute symptoms are at all diminished, the ointment is applied. In less severe cases the ointment is used from the first, with the aids of good diet, regular exercise, and tonics. All forms of counter-irritation are rigidly abstained from. The ointment is the citrine ointment of the United States' Pharmacopœia, but made with cod liver oil instead of lard and neat's foot oil. The first product is granular, but being kept over the fire, and thoroughly melted, and then stirred until cold, a perfectly smooth, soft homogeneous ointment, of a rich mahogany brown, is obtained.*

ON THE MECHANISM OF THE PRODUCTION AND DEVELOPMENT OF POSTERIOR STAPHYLOMA, AND ITS CONNEXION WITH INSUFFICIENCY OF THE INTERNAL RECTI.

Memoir presented to the Imperial Academy of Medicine, Nov. 27th, 1866. *By Dr. Giraud-Teulon. (Annales d'Oculistique, Nov. and Dec.,* 1866.)

It is well known that confirmed myopia is accompanied, among other symptoms, by two anatomical characteristics: 1. Progressive atrophy of the choroid; 2. Ectasia of the posterior coats of the eyeball, known as *posterior staphyloma.*

The myopia itself, which depends upon an elongation of the antero-posterior axis of the globe, is, in reality, a consequence of the ectasia.

It is intended, in the sequel, to study the mechanism which produces this ectasia, this elongation of the eyeball, this gradual change of its shape from a sphere to an ovoid. For this purpose

* The United States formula is, "Hydrargyri ʒi, Acidi Nitrici f. ʒxi, Adipis ʒiỹ, Ol. Ped. Bov. ʒix." Dr. Williams does not refer to the great value of the compressive bandage in phlyctenular cases. Whatever treatment is adopted, the bandage should not be omitted.

we have first to consider the anatomy of the part of the eye in which the change commences.

The sclerotic, at the posterior region of the eyeball, is composed of two layers, separated by a delicate, but appreciable, zone of connecting tissue, which forms a somewhat narrow disc around, and a little posterior to, the lamina cribrosa. The neurilemma of the optic nerve is composed of two layers like those of the sclerotic, also separated by connecting tissue. Of these layers, the internal becomes intimately blended with the lamina cribrosa, with the choroid, and with the inner layer of the sclerotic. The exterior layer of the neurilemma blends with the exterior layer of the sclerotic, and the intervening cellular tissue is continuous from one to the other organ.

All observers have remarked that the beginning of posterior staphyloma is indicated ophthalmoscopically by the appearance of a small whitish crescent at the outer border of the optic disc, generally in the horizontal plane. This crescent gradually increases in width along the horizontal meridian, its points embrace an ever-increasing portion of the disc, and it is ultimately met by a similar crescent, commencing on the inner border. The two become blended, and form an elliptic or circular zone around the point of penetration of the optic nerve.

Pathological anatomy then discovers that this zone is formed by the gradual distension, thinning, and, finally, atrophy of the choroid and the inner layer of sclerotic. The outer layer of sclerotic ultimately yields to pressure and recedes, forming a vesicle or distinct elevation. At the same time with these occurrences the eyeball becomes unduly tense. Sometimes the retinal veins pulsate spontaneously, and are more or less engorged or varicose. Lastly, the eyeball assumes a form manifestly ovoid, its long axis being antero-posterior.

This ectasia, or posterior staphyloma, with the myopia that it occasions, are generally the results of prolonged employment of the eyes about near objects. Hereditary myopia in young children, who have never exerted the eyes in near vision, is never accompanied by ectasia, although the latter condition is speedily produced when the eyes are so exerted.

We have therefore to determine what is the connection between exertion of the eyes upon near objects, and the production of a certain anatomical change. In the first place, in near vision what forces are called into play? Manifestly two, accommodation and convergence.

The myopic eye, however, has very little need of accommodation, and would rather relax than exert it. It remains to examine the phenomena of convergence.

The eyeball is suspended in equilibrium between two groups of muscles, the recti and the obliqui. Whether at rest or in motion this equilibrium is regulated by a law which has for its

object the maintenance of the spheroidal form of the globe. Upon this the accuracy of vision depends. When the eyeball moves in any direction, the muscles that contract to accomplish the movement would increase the internal pressure of the globe, and would thus distort its shape, if their antagonists did not relax in proportion. This principle, which must be borne carefully in mind, was laid down by M. Jules Guérin.

In simple convergence, on a horizontal plane, the axis of movement is the vertical axis of the globe, and the muscle in contraction is the internal rectus. The fulcrum is the surface of the spherical lever that is set in motion.

This granted, we may see that the contraction of the internal rectus tends to draw backwards its point of scleral insertion (and would therefore, if alone, simply pull back the whole eyeball). The resistance of the obliqui and of the rectus externus keeps the eyeball in its place, and compels it to turn upon its axis, producing adduction of the cornea. In this movement the point of insertion of the obliqui moves in an opposite direction, that is, from behind forwards, and from within outwards, for the same distance that the insertion of the internal rectus moves inwards and backwards. The insertion of the external rectus makes an equal movement forwards and inwards. Moreover, in proportion to the degree of convergence, the globe, by reason of its shape, tends to stretch the obliqui; an action that must manifestly be attended by an equal reaction on their parts, increasing internal pressure. The simple physiological movement of convergence on a horizontal plane necessarily produces therefore a tendency to increased intraocular tension. This tendency is much more manifest when the movement of convergence, as in most of the occupations of civilized life, is in a plane directed from above downwards, and from behind forwards. In § 9 of *Leçons sur le strabisme et la diplopie*, I have analyzed the influence of the elevation or depression of the plane of convergence upon the degree of intraocular tension.

When, for distant vision, the optical axis of one eye is directed downwards and inwards, the vertical meridian of that eye is inclined from above downwards, and from outside inwards. The other eye is parallel with the former, and no exaggerated tension is experienced. In *mutual* convergence it is otherwise: the two meridians remain vertical, and the inclination above mentioned does not occur. In the latter case, therefore, there is a muscle that restores or maintains, on each side, the verticality of the plane of the vertical meridian, and this can only be done by the superior oblique. In convergence upwards, it would be done by the inferior oblique. This special action of the obliqui is highly important, and necessarily increases their tendency to compress the globe; so that, in physiological convergence of the optic axes, we have all the conditions likely to increase the tension of the globe.

Since, however, all civilized eyes are not myopic, we must admit the necessity for some further condition, in order to change the tendency into an accomplished fact. Suppose that the circle described by the obliqui around the globe were a little more distant from the centre than in the normal state, we should then have an evident source of aggravation of the tendency. The farther the circle of the obliqui from the centre, the smaller that circle, and the more the pressure would be increased by adduction of the cornea.

A simple backward movement of the insertion of the obliqui is therefore of itself a dangerous source of increased intraocular pressure, and such an insertion is neither more nor less than the source of the preponderance of the muscles of abduction over those of adduction, of the external over the internal recti, or, as it is commonly called, of *insufficiency of the internal recti.*

Three conditions, of great importance as regards intraocular pressure, may therefore be combined in mutual convergence, two physiological, a third abnormal. The former are—1. The regular increase of the diameters of the circles that should be surrounded by the obliqui; 2. The excess of action imposed upon the superior obliqui in order to maintain the verticality of the axis of rotation of the globe. The abnormal condition is the insufficiency of the internal recti, which directly and rapidly increases the effects of the preceding.

It has now been shewn, therefore, that insufficiency of the internal recti dangerously increases the natural pressure consequent upon convergence. We have still to inquire why this increase is followed by ectasia or staphyloma posterior, and not by excavation of the optic disc as in glaucoma.

The matter is very simple. In glaucoma the excessive pressure originates within the globe, and acts uniformly on all points of its periphery. Doubtless it is the same in the case under consideration, and the pressure from within outwards is the same at all points. This pressure has, however, a point of departure in the ocular membranes. It would not exist unless the sclerotic were pulled from behind forwards, and from within outwards, at the place of insertion of the obliqui, by the very fact of convergence. We therefore have the highly elastic sac of the choroid compelled to assume the form of an ovoid, with its long axis directed forwards and backwards (the direction in which the coats of the eye are the least supported), while the external coat, the sclerotic, is drawn forwards. If we reflect on the anatomical arrangement by which the choroid and the lamina cribrosa, and the internal layer of the sclerotic, are intimately united, it is easy to see that the two layers of the sclerotic will become separated in the region where their union is a matter of simple contact: the region where lax connecting tissue surrounds the circle in which the sclerotic blends with the optic neurilemma. The

scleral boundary of the optic disc will follow the movement of the choroidal boundary from before backwards; while the external layer of the sclera is drawn from behind forwards, and from within outwards. The two layers of the sclera are thus dissociated, and made to glide one over the other. Such appears to be the order of events which superadds posterior staphyloma to insufficiency of the internal recti, when that insufficiency is brought into prominence by the need for binocular vision at short distances.

The opinions thus stated are not merely speculative, but are based upon clinical observation.

There is no ophthalmologist who has not met with persons presenting more or less evident posterior staphyloma, without myopia. Donders himself, to whom is due the belief that staphyloma is pathognomonic of myopia, has observed, even frequently, slight traces of atrophy at the external margin of the disc, and sometimes a complete ring, not only without myopia, but in two cases of hypermetropia. The same kind of experience has directed my attention to the matter, and I have been able, in the course of 18 months, to observe thirty or forty cases that throw new light on this important question. The appended table contains some details of thirty-eight cases, carefully observed, comprising eight of slight myopia, that is, less than $\frac{1}{18}$, and mostly about $\frac{1}{30}$, or $\frac{1}{36}$, twenty of emmetropia, and ten of hypermetropia. Among the eight myopic cases, the myopia was manifestly acquired in two, and in two others it only affected one eye.

In all the patients the external border of the optic disc presented traces more or less marked, but undeniable, of choroidal atrophy or commencing posterior staphyloma. All the patients presented symptoms of insufficiency of the internal recti; demonstrated, after the manner of v. Graefe, by a prism with its angle superior or inferior. The facts set forth in the table, together with the circumstance that myopia of a high degree may exist without staphyloma, appear to justify the following conclusions:—

1. If myopia may generally be referred to the existence or the development of ectasia as its immediate cause, this ectasia is itself the result of causes immediate and remote, or predisposing. The first is employment of the eyes upon near objects; the second insufficiency of the internal recti.

2. Among the diagnostic signs of insufficiency of the internal recti, and as one of the principal of them, it will be necessary to place, for the future, the presence of posterior staphyloma, and especially the presence of slight wasting of the choroid at the outer border of the optic disc. In a word, posterior staphyloma is rather a symptom of insufficient convergence than of myopia; or, rather, myopia and staphyloma posterior indicate insufficiency of the internal recti.

Table of Cases.

No.	Description.	Symptoms.	Refraction.	Remarks.
1	Mlle. X., æt. 16, Pupil Teacher.	Muscular asthen. Insufficiency of Rectus int.	R. Myopia $\frac{1}{36}$. L. Myopia $\frac{1}{24}$.	Double crescent; choroid hyperæmic.
2	M. X., Commercial Traveller.	Recurrent conjunctivitis. Insufficiency of R. int.	Acuity and range normal.	Staphyloma posterior.
3	M. X., Magistrate.	Muscular asthenopia.	Ditto.	Ditto.
4	Mlle. L., æt. 15.	Asthenopia.	Slight latent H. Apparent M.	Ditto.
5	Mlle. C., æt. 16.	Musc. asth. Bleph. cil. Insuff. of R. int. = 3°.	Acuity and range normal.	Staphyloma and choroidal hyperæmia.
6	Mlle. M., æt. 13.	Musc. and accom. asthenopia. Ins. R. int.	Slight H.	Staph. post.
7	M. M., æt. 36, Merchant.	Complex asthen.; nervous affection; insufficiency both of convergence and divergence.	H = $\frac{1}{11}$.	Ditto.
8	Mlle. M. T., æt. 10.	Double musc. and accom. asthenopia. Insuff. of R. int., amounting to external strabismus.	H = $\frac{1}{3}$.	Ditto.
9	M. D., æt. 29, Naval Officer.	Asthen. Paresis of accom. Insuff. of R. int. = 6° or 8°.	R. M = $\frac{1}{36}$. L. Normal.	Ditto.
10	M. M., æt. 67.	Diminished acuity; no lens useful. Insuff. of R. int.	Emmetropic.	Ditto. Tension. Venous pulse.

11	M. J., æt. 30.	Crossed diplopia. Insuff. of R. int. to ext. strabismus.	Ditto.	Double staph. post.
12	M. L., æt. 6½.	Intermitt. strab. diverg., or insuff. R. int.	Ditto, or very slight H.	Ditto. Choroidal hyperæmia.
13	Mme. M., æt. 46.	Amblyopia R. Apoplexy of macula lutea. Evident insuff. of R. int., notwithstanding the absence of binocular fixation.	L. Manifest H. = $\frac{1}{36}$.	Ditto.
14	M. B. B.	Mydriasis L. Asthenopia. Insufficiency = 6°.	M. = $\frac{1}{24}$ or $\frac{1}{30}$.	Ditto.
15	Mme. B., æt. 55.	Musc. Asthen. Insuff. of R. int.	Emmetropic.	Ditto. Venous pulse.
16	M. C. L., æt. 19.	Musc. Asthen. Insuff. = 8°.	Slight M. Manifestly acute.	Double staph. post.
17	M. S., æt. 60.	Bleph. chron. Insuff. of R. int.	M. = $\frac{1}{30}$.	Ditto.
18	M. F., æt. 65.	Asthen. Insuff. of R. int.	Emmetropic.	Ditto.
19	M. C., æt. 30.	Ditto.	Acuity and range normal.	Ditto.
20	Mme. V. M.	Ditto.	Manifest H. = $\frac{1}{48}$.	Ditto.
21	M. T.	Bleph. cil. chron. Insuff. of R. int.	Acuity and range normal.	Ditto.
22	Mme. X., æt. 66.	Asthen. Insuff. of R. int.	Manifest H. = $\frac{1}{24}$.	Ditto.
23	Mme. H., æt. 47.	Ditto.	Emmetropic.	Ditto.
24	Mme. B., æt. 45.	Vertiginous asthenopia. Insuff. of R. int.	R. Myop. = $\frac{1}{24}$. L. Emmetropic.	Ditto. Venous pulse.

TABLE OF CASES—*continued.*

No.	Description.	Symptoms.	Refraction.	Remarks.
25	D., æt. 14.	Musc. asthen. Insuff. of R. int.	L. Emmetropic. R. Myop. = $\frac{1}{15}$.	Double staph. post. Venous pulse.
26	Mme. X., æt. 75.	Ditto.	L. Acquired H. = $\frac{1}{30}$. R. H. = $\frac{1}{20}$.	Ditto.
27	Mme. X., æt. 48.	Ditto.	Acquired M. = $\frac{1}{36}$.	Ditto.
28	M. B., æt. 29.	Hemeralopia. Strabismus diver. = $1\frac{1}{2}$ line. Peripheral field of vision intact.	Acuity normal M. = $\frac{1}{36}$.	Do. Tension increased.
29	M. Dr. G.	Asthen. Insuff. R. int.	R. Emmetropic. L. Manifest H. = $\frac{1}{30}$.	Increased tension.
30	Mme. H.	Ditto.	Acuity and range normal.	Staphylomatous crescent.
30B	Her son.	Ditto.	R. Myop. = $\frac{1}{12}$. L. Myop. = $\frac{1}{36}$.	Ditto.
31	..	Ditto. Eyeball hard.	Acuity and range normal.	Ditto. Also with internal crescent.
32	Mme. P., æt. 53.	Asthen. Insuff. R. int.	Manifest H. = $\frac{1}{30}$.	Staphylomatous crescent.
33	Mlle. J., æt. 26.	Confirmed asthenopia. Divergent strabismus.	R. H = $\frac{1}{8}$. L. Amblyopia.	Ditto.
34	Mlle. L., æt. 8.	Asthen. Insuff. of R. int.	Emmetropic.	Ditto.

35	M. D.	Muscæ volitantes. Insuff. of R. int. *Corrected by prisms.*	Emmetropic.	Ditto.
36	M. X.	Acuity reduced to $\frac{1}{2}$. Insuff. of R. int.	Myopia = $\frac{1}{18}$.	Ditto.
37	M. D., æt. 35.	Asthenopia. Insuff. of R. int., amounting to strabismus.	R. Emmetropic. L. Image suppressed.	Ditto.
38	M. V., æt. 70.	Manifest insuff. of R. int.	Myopia = $\frac{1}{38}$.	Choroido retinitis, atrophy, large posterior staphyloma.
39	M. H.	Very well marked muscular asthenopia.	H. = $\frac{1}{18}$.	Nothing noticed.

P.S.—The foregoing paper was already finished, when I had the pleasure of hearing the views of Prof. v. Graefe. In a lecture with which he honoured our clinique, M. de Graefe laid down propositions between which and the foregoing there is a close connection. (See page 370.)

The learned Berlin Professor attributes the progressive increase of myopia to an insufficiency of convergent force, or to a dynamic divergent strabismus; and is sufficiently convinced of the accuracy of this view to propose and employ, as a single means to check the progress of posterior staphyloma, the remedy for divergent strabismus, tenotomy of the external rectus. The preceding pages justify the statement that I was about to enter upon the same path; and I have now a case of extreme myopia, arrived at the period of intermittent divergent strabismus, in which tenotomy of the external rectus suddenly diminished the myopia by $\frac{1}{13}$, or from $\frac{1}{5}$ to $\frac{1}{8}$.

It is only just to recall here the attempts made in 1841, by Bonnet, Cuvier, Philips, Jules Guérin, and others, to apply tenotomy to the treatment of myopia. The results then obtained were questioned; but the present knowledge of ocular refraction will make new trials in the same direction more satisfactory.

A MODIFICATION OF THE STRABISMUS OPERATION.

By Dr. R. Liebreich (*Arch. f. O.* xii, 2, p. 298).

By the present methods of operating for squint, it is possible to obtain a correction of $2'''$ or $2\frac{1}{2}'''$ in adults, of $2\frac{1}{2}'''$ or $3'''$ in children. If the deviation be more than this, two, three, or more operations will be required. The division of the necessary correction between two operations, one for each eye, is productive of certain advantages with regard to symmetry and equality of movement; but the repetition of tenotomy on a muscle that has been previously divided is attended by many evils. It is impossible to foresee whether the effect produced by a second operation will be small or great. The cicatrix of the former section may be imperfectly divided, and may entirely nullify the effect of the subsequent one; or, on the other hand, the complete division of such a cicatrix may require a very extensive operation, which may be followed by divergence and loss of movement, sinking of the caruncle, and all the bad results of the old and now abandoned methods of operation. Urged by these considerations, and desirous to improve the accepted methods of tenotomy, Dr. Liebreich, 18 months ago, entered upon a careful study of the anatomy of the parts concerned.

The ocular capsule, which surrounds the whole eyeball with the exception of the cornea, is composed of two widely different

portions. The posterior portion forms a cup, with a firm smooth internal surface, in which the eyeball moves freely, like the head of a bone in its socket. This cup is perforated by the four recti muscles, and forms, at the line of perforation, a sharply defined ring, so closely adherent to the muscles, that no movement can take place between them. The union between the muscles and the posterior part of the capsule is further strengthened by vaginal processes, which spring from the outside of the capsule, and cover the muscles for some distance in the orbit. Towards the eyeball there are no sheathing processes; but the posterior part of the capsule terminates abruptly at the circle of the muscular perforations; and the muscles, anterior to this circle, are for a short distance completely free from sheaths. Before their tendons reach the sclera, they pass between that membrane and the anterior part of the capsule, and become united to the latter. This anterior portion of the capsule bears to the posterior portion somewhat the relation of a convex cover to a bowl. It is much thinner than the posterior portion, and is very difficult of dissection, since, like the conjunctiva, it rapidly loses its firmness after death.

Following the anterior portion from the anterior pole of the eyeball, we begin with a circular opening the size of the cornea, through which the latter projects. The anterior margin of this opening is united to the sclera; and, in a zone limited by this margin in front, and by the insertions of the recti muscles behind, the sclera, capsule, and conjunctiva are almost immovably adherent. Beyond the boundary of this zone the conditions change. The union between capsule and sclera is broken by the intervention of the muscles; and some loose irregular connecting tissue that here exists between the several structures has possibly furnished the basis of such complicated and fantastic descriptions as those of Guérin. The sheaths said to invest the muscles from their perforation of the capsule to their insertion, and that have been used to explain many matters, have no existence. On the contrary, as already described, the anterior half of the capsule rests on the anterior surfaces of the muscles, and is closely united to them; while the conjunctiva is closely united to the capsule over an irregular zone, the boundary of which, in excentric movements of the eye, is indicated by a wrinkle. Beyond this boundary the union between the capsule and the conjunctiva is much looser. Part of the tissue composing the anterior portion of the capsule is reflected into the eyelids; and the rest is applied to the margin of the posterior portion, which itself sends prolongations to the margin of the orbit.

The foregoing description contains three points of special importance in the squint operation.

1. The union between the muscles and the ocular capsule is double. There is first the circular union between the posterior

part of the capsule and the muscles, strengthened by the sheathing processes. There is secondly the union between the anterior part of the capsule and the muscles beneath it.

2. The conjunctiva is firmly adherent to the anterior portion of the capsule, from the margin of the cornea to the outer edge of an irregular zone. It is therefore indirectly in very intimate connection with the muscles.

3. The caruncle and plica semilunaris rest upon a band that passes from the capsule to the margin of the orbit. The contraction of the rectus internus, in adduction of the eye, stretches this band, and draws with it the caruncle towards the inner wall of the orbit. At the same time the outer edge of the caruncle, and the portion of conjunctiva next to it, are pulled backwards into a wrinkle. This is partly because the conjunctiva moves with the eyeball up to the boundary of the line of adhesion; partly because the muscle, on account of its union with the anterior portion of the capsule, draws this backwards in contraction.

It follows from the first of these three conditions that a displacement backwards of the insertion of a rectus muscle can only be obtained by a displacement backwards of the part of the anterior capsule that covers the muscle. This capsule, if not divided, would retain the severed tendon in its original position. On account, however, of its close adhesion to the muscle it would be very difficult, even if possible, to leave it uninjured; and, even in subconjunctival tenotomy, the capsule must usually be divided to the same extent as the tendon itself. This division of the capsule with the tendon explains how it is that a displacement backwards of the part of the anterior capsule that covers the tendon, as well as of the ring of posterior capsule that is adherent to the muscle, and therefore a displacement of the point of insertion itself, is actually effected. By enlarging the section of the capsule (v. Graefe's separation of lateral processes) the displacement backwards may be somewhat increased, but the second of the three points above mentioned is an obstacle to any considerable increase. The union between conjunctiva and capsule forbids any great yielding of the latter, unless the former also be freely divided. If this be done, we see the effect of the third point. On account of the union between muscle, capsule, and caruncle, the displaced muscle draws the caruncle, plica semilunaris, and divided conjunctiva, so much backwards and inwards that these parts assume, when the patient looks straight forward, the position that is natural to them during extreme adduction. The distance between the plica and the inner margin of the cornea is increased, the portion of sclerotic visible at the inner side of the eyeball is enlarged, and the eye assumes the deformity of aspect characteristic of the old methods of operation. To obviate these evils, and to obtain any desired amount of result from a single operation, Dr. Liebreich proceeds as follows:—

For tenotomy of the internal rectus he lifts with forceps a fold of conjunctiva over the lower end of the insertion of the muscle, and divides this fold with scissors. He then carries the scissors through the wound, between the conjunctiva and the capsule, and carefully separates these structures as far as the plica semilunaris. The plica and caruncle are also to be separated in the same manner from the parts beneath them. After thus rendering the portion of capsule to be divided entirely free from the superficial structures, the tendon is taken up and cut through in the ordinary manner, and the perpendicular division of the capsule is enlarged, upwards and downwards, to an extent corresponding with the amount of displacement that is desired. Finally, the conjunctival wound is brought together by suture.

For the external rectus a similar operation is performed, in which the conjunctiva must be separated from the parts beneath as far as to the point that is drawn directly backwards in extreme abduction.

For these proceedings Dr. Liebreich claims the following advantages over former methods:—

1. *A greater freedom and a far greater range of effect.*
2. *Avoidance of a depressed caruncle or an unsightly cicarix.*
3. *Avoidance, in every case, of more than two operations, that is, of more than one on each eye.*

With regard to the first of these, it is possible to obtain either the effect of an ordinary tenotomy or a correction of 4‴ or more in adults, and of 5‴ or more in children. Dr. Liebreich disclaims, however, the practice of tenotomy on one eye only in very extreme squint, and follows v. Graefe in preferring to divide the effect between both eyes. It is only in exceptional cases in which the mobility inwards of the squinting eye is much increased, and the opening of the lids not wider than in the other eye, that he rectifies a very large deviation by a single tenotomy. Under other circumstances, as when the departure of the patient, or some other reason, prevents a second operation, he prefers an unilateral modified tenotomy to the practice followed, in such cases, at Moorfields and elsewhere, of dividing both internal recti at the same time. He more frequently is satisfied with a single tenotomy when the deviation does not exceed 3‴ in adults. or 4‴ in children. He attaches especial importance to the conjunctival suture, for which he uses a fine curved needle and the finest English black silk. Even if several stitches are inserted, no trace of a scar will remain.

Dr. Liebreich considers the third point of advantage, the rendering repeated operations unnecessary, to be the most important. He states that in this modified tenotomy, especially when the section of capsule is extensive, the difference between the immediate and the ultimate result is very great; and that, where the mobility inwards of the eye is at first almost destroyed,

it will, nevertheless, be completely restored in the course of time.

AMAUROTIC FORM OF DOUBLE QUOTIDIAN INTERMITTENT.

By Dr. Testelin (*Bull. Méd. du Nord*, 1866, p. 221; *Annales d'Omlistique*, vol. 56, p. 317).

Dr. Testelin relates the case of Marie K., æt. 10 years, brought to him on the 2nd of July, 1866. Her father stated that she became periodically blind of the left eye, which, beyond a little redness of the palpebral conjunctiva, presented no morbid appearance. Its vision was equal to that of the right, its pupil regular and contractile, and the ophthalmoscope revealed nothing abnormal. The child was very intelligent, and it appeared from her statement and replies that she was attacked twice a day, about 11 a.m. and about 4 p.m., with complete temporary blindness of the left eye. Each attack was ushered in by slight *malaise*, a kind of shiver, and a little headache; then, all at once, the left cheek became warmer, the left eye tingled a little, and vision ceased completely, without any appearance of sparks or bright lines. The patient was very positive on this point, and states that when the right eye was closed she was in absolute darkness. This condition lasted from fifteen minutes to half an hour. The child lived in a house close to a water-course recently covered, she attended a day school on the bank of the Lower Deûle, and went frequently into a field also on the bank of the Deûle.

The diagnosis of malarious fever was clear, and Dr. Testelin prescribed 30 *grammes** of sulphate of quinine, to be taken daily in a single dose. On July 3rd the attacks were already modified, only the morning paroxysm occurred, and that only lasted four minutes. 4th. The morning attack lasted ten minutes, and the afternoon one fifteen minutes. 5th. No attack. Two grammes of sulphate of quinine and one gramme of tartaric acid, in 40 pills, eight to be taken in one dose daily. 6th. The morning attack consisted only of a slight obscurity; the afternoon attack was a quarter of an hour before its usual time, and lasted a quarter of an hour (8 pills). 7th. No morning attack. At 4 p.m. loss of vision for 20 minutes (8 pills). 8th. No attack (6 pills). 9th. Ten minutes blindness at 10h. 10m. a.m. (10 pills). 10th. No attack (10 pills). From this day the attacks ceased, but the patient took, on the 11th, 10 pills, on the 12th 8 pills, on the 13th 6 pills, the 14th 4 pills, the 15th 2 pills.

The patient then went for a fortnight to a healthy country

* Qy. *centigrammes*.

neighbourhood, and took carbonate of iron in full doses for a month. She has since continued free from the disorder, and in perfect health.

THE TREATMENT OF FISTULA OF THE CORNEA.

By Dr. Wecker. El Papellon Médico (of Madrid); Annales d'Oculistique, vol. 56, p. 305.

El Papellon Médico publishes a series of letters between Drs. Wecker and Delgado upon interesting points of ophthalmic practice. Dr. Wecker writes about corneal fistula as follows:—

It sometimes occurs that a corneal ulcer perforates that membrane, or that an abcess in its substance opens both internally and externally, leaving a permanent orifice through which the aqueous humour escapes. Generally, prior to the perforation, the membrane of Descemet protrudes through the opening in the corneal tissue, and ultimately the apex of this protrusion bursts, and the membrane tears into little flaps, which may be carried forwards by the outflow of aqueous humour, so as to meet, and become adherent to, the anterior boundary membrane (Bowman's) of the cornea. In this way a permanent fistula may be formed, analogous to a fistula uniting two mucous tracts. Attempts have been made to close such openings by the free use of solid nitrate of silver, but often without success; and, when successful, at the cost of a large and indelible cicatrix. Iridectomy, the strangulation of a portion of iris in the fistula, and a crucial incision of the walls of the opening with a paracentesis needle, have all been recommended; but such operations are very difficult in an eye with no anterior chamber, and softened by the loss of its aqueous humour.

Having frequently seen a corneal fistula established after the rupture of a protrusion of the membrane of Descemet, and convinced that the fistula is due to the eversion of the portions of that membrane, M. Wecker has devised the following method of treatment. He introduces into the opening a very fine straight forceps, with smooth points, and seizes the wall of the fistulous track, so as to bruise or tear the lining of the track, and to denude the proper corneal tissue. During this proceeding, if the fistula be central, the smooth points of the forceps come in contact with the anterior capsule of the crystalline lens, so that great care and gentleness are required. Having nipped or squeezed the wall of the fistula at several points, a strong solution of atropine is applied, and a compressive bandage is worn for from eight to fifteen days, being removed once daily for the repetition of the atropine.

By this method M. Wecker has recently cured a case of corneal fistula that had existed for ten months notwithstanding various treatment. The resulting cicatrix was smaller than it would have been after the use of nitrate of silver.

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